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THE ETIOLOGY OF LUNG ABSCESS

A Clinical Analysis of 447 Cases Occurring at the Boston City Hospital from 1926 to 1935, Inclusive

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THE etiology of lung abscess has become a matter of special concern to otolaryngologists, particularly because of the fact that tonsillectomy has been found to be the background of a large percentage of these cases in most of the large clinics. The literature is replete with such reports showing percentages varying from 10 to 40 per cent or more. Lord[1] in a series of 227 cases found that in 22 per cent the abscess developed as a sequel of tonsillectomy. Hedblom[2] in a total of 692 lung abscesses found 31 per cent to follow tonsillectomy. Schlueter and Weidlein,[3] in a series of 1908 cases attributed 15 per cent to tonsillectomy. Pinchin and Morlock[4] in England, show that 12 per cent of their 25 cases of lung abscess followed tonsillectomy. It is notable that, although the percentages vary considerably, in the series containing the largest number of abscesses the incidence of those following tonsillectomy is relatively low. Similarly Moore,[5] in a questionnaire to otolaryngologists all over this country, gathered reports of 450,000 tonsillectomies, 202 of which were followed by lung abscess — a ratio of 1:2200. Crowe and Scarff,[6] of the Mayo Clinic, recorded 3500 tonsillectomies, not one of which was followed by lung abscess. This is in agreement with Moore's finding that the larger the number of tonsillectomies performed, the smaller the percentage of lung abscess as a complication. Added to this fact is the outstanding one that most otolaryngologists encounter lung abscesses only occasionally. Apparently no attempt has been made to determine the incidence of lung abscess following operations on the upper respiratory tract, or to compare such a figure with that following other operative procedures or medical conditions such as pneumonia.

Maxwell[7] in a study of 315 autopsies following deaths from lung abscess at St. Bartholomew's and the Royal Chest Hospital, in London, analyzed all the causes, and found a small number of lung abscesses following operations on the upper respiratory tract in comparison with all other causes (Table 1). Although this series represents only cases which failed to survive, the figures are of interest because they are very much the same as those found in the foreign literature covering the incidence of all types of lung abscess regardless of outcome.

This table shows how much more important a role pneumonia and abdominal operative procedures play in these cases than do operative conditions involving the upper respiratory tract. This being so, it seemed to me that a study of all lung abscesses, of both medical and surgical origin, occurring in a large clinic such as that of the Boston City Hospital over a ten-year period, would be of value in classifying this complication, and might produce some interesting and important data. This clinic has served the purpose especially well because it cares for a large clientele of indigent individuals who have little means for paying ward rates or professional fees. As a result, not only do patients with serious postoperative complications return to the hospital, but others operated on elsewhere come there for the treatment of long-drawn-out complications, which would otherwise be expensive.

The period from 1926 to 1935, inclusive, was chosen for this investigation. All lung-abscess cases were abstracted, studied and classified. Similarly, all the operative procedures carried out during this period were totaled, so that the ratio of operations to lung abscess could be determined.

A total of 447 cases in which the diagnosis of lung abscess was at any time made were abstracted, and all proved lung abscesses were selected for comparative study. X-ray pictures were examined, and histories and autopsy reports were studied.

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The total of lung-abscess cases accepted for investigation was 276. These were then separated into those of postoperative origin and those with other causes; 196 cases were of medical origin, 76 occurred as a postoperative complication and 4 were a result of trauma.

Table 1. *Causal Conditions in Single Lung Abscess (199 cases), as Reported by Maxwell.*

LESIONS OF THE RESPIRATORY TRACT:	
Oropharynx:	
Operation for carcinoma of tongue	2
Tonsillectomy	2
Operation for carcinoma of tonsil	1
Larynx:	
Operation for carcinoma	2
Trachea:	
Tracheotomy	3
Bronchi:	
Foreign body	2
Bronchiectasis	2
Carcinoma	32
Perforating carcinoma of esophagus	13
Lungs:	
Lobar pneumonia	19
Bronchopneumonia	24
Actinomycosis	2
Suppuration of hydatid cyst	1
Secondary carcinoma	2
Chest:	
Injury	2
Operation for empyema	2
ABDOMINAL LESIONS:	
Subphrenic abscess (operation)	3
Perforated gastric and duodenal ulcer (operation)	7
Gastroenterostomy	6
Gastrectomy	1
Bleeding gastric ulcer (operation)	1
Cholecystectomy	3
Appendix abscess (operation)	4
Umbilical hernia (operation)	1
Strangulated hernia (operation)	1
Carcinoma of colon (operation)	3
Hysterectomy	1
Radium for carcinoma of cervix	1
Bladder and prostate (operation)	6
SEPTIC LESIONS ELSEWHERE:	
Cutaneous sepsis	4
Puerperal sepsis	4
Pneumococcal septicemia	1
Osteomyelitis	5
Sepsic arthritis	1
Otitis media	4
Lateral sinus thrombosis	3
Perinephric abscess	1
LESIONS OF THE CENTRAL NERVOUS SYSTEM:	
Injury to skull or brain	3
Vascular lesion	1
Spinal injury	1
MISCELLANEOUS CAUSES:	
Diabetes mellitus	2
Nonperforating carcinoma of esophagus	3
Liver abscess (doubtful cause)	1
CAUSE NOT EVIDENT:	
Primary group	16

Table 2 represents all lung abscesses occurring in patients who had not been operated on. The 57 cases of chronic cough included bronchitis, asthma and bronchiectasis, and a number of factors, such as arteriosclerosis, cardiac disease and alcoholism, were involved. In these no definite history of pneumonia or influenza as a background could be elicited. Undoubtedly a fair percentage could have been traced back to the common cold, influenza or some acute process, with intervening atelectasis. In studying these abstracts it became evident that in the latter half of the decade there was a gradual realization on the part of physicians of the role that pulmonary atelectasis plays in stagnation of infected material in shut-off portions of the lung.

Of the 196 cases of medical origin, 189 were

Table 2. *Lung Abscesses in 200 Unoperated Cases.*

MEDICAL CAUSES:		**CONTRIBUTING CAUSES:**	
Pneumonia	104	Alcoholism	5
Acute abscess	59	Diabetes	5
Chronic abscess	45	Pleurisy and empyema	29
Chronic cough (Indefinite history; a number of contributing factors)	57	Syphilis	5
		Industrial (sandpaper worker)	1
Influenza	15	Atelectasis in one or more lobes	17
Tuberculosis of lung	10	Cardiac disease and arteriosclerosis	11
Carcinoma of lung	3		
Carcinoma of tongue	1		
Subphrenic abscess	2		
Acute sepsis	4		
Total cases	196		
TRAUMATIC CAUSES:			
Inhalation of foreign body			1
Accident and fracture of jaw and femur			3
Total cases			4

solitary abscesses involving one lobe of either lung, and 7 were multiple abscesses, including either abscesses in different lobes or more than one abscess in a single lobe (Table 3). From these

Table 3. *Location of Lung Abscesses of Medical Origin, Solitary and Multiple (196 cases).*

RIGHT LUNG		LEFT LUNG	
Upper lobe	38	Upper lobe	30
Middle lobe	19		
Lower lobe	75	Lower lobe	48
Totals	132		78

figures it is evident that the right lung and the lower lobes were more commonly affected than the left lung and the upper lobes. Almost all the abscesses following an acute process, with the exception of those due to acute sepsis and those following accident, were solitary. The multiple abscesses were those of chronic background. It is quite possible that there were more cases of multiple abscess than were diagnosed as such. Some of the cases which came to autopsy were found to have multiple abscesses, although during life these abscesses were diagnosed as solitary. For that reason the solitary and multiple abscesses were consolidated in one table, although only 7 cases of this series were proved to have multiple abscesses.

LUNG ABSCESS OF MEDICAL ORIGIN

Examination of all cases of lung abscess following medical conditions disclosed certain definite common factors.

A great number of these patients were in poor general condition. Some had diabetes, arteriosclerosis or cardiac disease, and a number were

victims of alcoholism. Unclean mouths and bad teeth and gums were present in nearly every case. Some patients had chronic lung trouble, such as bronchitis, asthma or bronchiectasis. All these conditions represented fertile soil for active lung infection. Supervening upon this and acting as a precipitating cause, were influenza and pneumonia. With the acute reaction incident to these infections, atelectasis to a smaller or larger extent took place, and the sequence of events was pneumonitis, necrosis and abscess formation.

The bacteriological findings in all these cases were not uniform. Anaerobes, streptococci, staphylococci, pneumococci, fusiform bacilli and spirochetes were present in various combinations. Even though an occasional case materially improved when arsphenamine was used, by and large this drug did not seem to affect materially the course of the disease. The claim that is sometimes made that spirochetes and fusiform bacilli are the important factors in the production of these abscesses was therefore not substantiated by our analysis. It would seem that this is not the only type of infection that is operative. The importance of the general condition of the patient, the virulence of the organisms, and the mechanical factors involved in atelectasis, which was probably present in a large percentage of the cases, cannot be overestimated in the production of abscess.

One is struck by the large number of cases following pneumonia or pneumonic processes. In this ten-year period there were 15,177 cases of pneumonia in the Boston City Hospital, and 104 cases of lung abscess following pneumonia, a ratio of 1:146. This complication was recognized by many earlier writers, among them Laennec,[8] Zenker[9] and Tuffier.[10] In fact, lung abscess was considered a fairly frequent sequel to pneumonia. But as increasing attention was focused on lung abscess as a postoperative complication, particularly following tonsillectomy, pneumonia and general respiratory infection received less and less serious consideration as causes. A contributory factor was the realization that the pneumococcus was frequently not the only organism present. With the advent and the great spread of modern surgery came a tremendous increase in postoperative complications, as a result of which pneumonic conditions per se receded as a cause of lung abscess. The present survey, however, again reveals that a very large proportion of lung abscesses are still due to acute and chronic pneumonic processes, and in all probability outnumber those occurring as postoperative complications.

POSTOPERATIVE LUNG ABSCESS

We now come to a consideration of the lung abscesses which followed operative procedures. There were 76 such cases in this series. In addition, 13 patients who had had tonsillectomies performed elsewhere were brought to the Boston City Hospital for the care of the pulmonary complications. These cannot be included in our figures in showing the relation between operative cases and ensuing lung abscess. Such cases are not unusual, because tonsillectomy patients usually leave the hospital within a day or two after the operation, whereas in almost all other cases the hospital stay averages ten days, during which lung abscess is likely to occur. It has therefore happened that the tonsillectomy was performed at one hospital and the postoperative complication was cared for at another. Furthermore, many patients with acute postoperative atelectasis, infarction, or pneumonia following abdominal operations die before a lung abscess can supervene. These cases also cannot be included in our figures, but offer food for serious speculation.

Table 4. *Postoperative Lung Abscesses (76 cases).*

OPERATIONS	
Tonsils and adenoids	16
Nose	2
Teeth	10
Hernia	4
Appendix	9
Kidney and bladder	3
Stomach	9
Gall bladder	9
Uterus and tubes	14
Total cases	76

Table 4 represents the distribution of our 76 cases of postoperative lung abscess. They were studied with the special purpose of finding, if possible, common factors as a background for the ensuing abscess. Most of these patients were in poor physical condition; the operations and anesthesias, except for the upper respiratory tract, were of long duration, and opportunities for complications were frequent.

The distribution of abscesses was greater for the right lung and lower lobes (Table 5), as was found in the cases following pneumonia or infection of the nonoperative type. This distribution is important because it is most typical of abscesses of aspiratory origin. On the other hand, those who favor embolus as a cause point to the fact that in proved embolic cases also the abscess distribution favors the right lung and lower lobes.

Table 5. *Location of Postoperative Lung Abscesses, Solitary and Multiple (76 cases).*

RIGHT LUNG		LEFT LUNG	
Upper lobe	9	Upper lobe	6
Middle lobe	9	Middle lobe	0
Lower lobe	35	Lower lobe	20
Totals	53		26

The total numbers of the more common operations performed at the Boston City Hospital during the years 1926-1935, inclusive, are given in Table 6, together with the cases subsequently de-

veloping lung abscess. More lung abscesses followed operation for removal of tonsils and adenoids than for any other operative procedure, but because of the large number of operations — twice that of any one of the others — the ratio of incidence (1:1654) was the lowest of all.

Table 6. *Total Number of Operations and of Subsequent Lung Abscesses at the Boston City Hospital (1926-1935, inclusive).*

	OPERATIONS	CASES OF LUNG ABSCESS	RATIO
Tonsils and adenoids	26,473	16	1:1654
Teeth	3,204	10	1:320
Appendix	12,702	9	1:1411
Stomach	486	9	1:54
Gall bladder	2,328	9	1:258
Uterus and tubes*	10,839	14	1:774

*Seven-year period, 1929-1935, inclusive.

Table 7 gives the total number of cases of lung abscess developing after operations on the upper respiratory tract and after abdominal operations.

POST-TONSILLECTOMY ABSCESS

Of the 26,473 tonsillectomies and adenoidectomies performed, 17,705 were on children and 8768 on adults. Two abscesses followed those performed on children, and 14 followed those on adults. General anesthesia was used for all the children. Eighty-five adults were operated on under local anesthesia, and the rest under general anesthesia; none of the former developed lung abscess. The ratio of lung abscess to operation in the 26,473 cases was 1:1654 — 1:8852 in children and 1:626 in adults. There was much variation from year to year in this ratio. In 1935 there were no lung abscesses and 4241 tonsillectomies. The last three years of the decade produced 5 lung abscesses, or a ratio to tonsillectomies of 1:2309. In the last five years there has been a greater realization of the need for careful observation, and as a result of more conscientious attention to preoperative condition and to methods of procedure, complications have been less frequent. Presumably because otolaryngologists always pay more personal attention to their private cases than they do to those in the hospital, the incidence of lung abscesses in such cases is low.

Table 7. *Postoperative Cases of Lung Abscess at the Boston City Hospital (1926-1935, inclusive).*

FOLLOWING OPERATIONS ON UPPER RESPIRATORY TRACT		FOLLOWING ABDOMINAL OPERATIONS	
Tonsils and adenoids	16	Hernia	4
Nose	2	Appendix	9
Teeth	10	Kidney and bladder	3
		Stomach	9
		Gall bladder	9
		Uterus and tubes	14
Totals	28		48

In our study of post-tonsillectomy lung abscesses, we examined the factors concerned in their production in the light of our experience with the methods used in this institution. We divided these into *extrinsic* and *intrinsic* factors. The former we considered those which did not depend upon the patient or did not concern his general condition. These were operative position, — upright or Rose, — the use of suction during operation, and preoperative medication. All other factors, classed as intrinsic, included the patient's preoperative condition, the kind of anesthesia employed, operative trauma and bleeding, and aftercare.

Among the extrinsic factors, the operative position was considered first. All our tonsillectomies were performed with the patient in the upright position, and suction was generally used to keep the throat clear. Since only 1 lung abscess occurred in 1654 tonsillectomies, a figure which compares favorably with those of other clinics where the Rose operative position is used, it is difficult to believe that operative position was an important factor. Likewise, the use of suction, which some observers fear may cause more bleeding, and thus indirectly inhalation and an increased risk of thrombosis, is in the main a negligible factor, because if it were not, since it was frequently employed, we should have had many more abscesses than are shown in this investigation.

Preoperative medication in our cases certainly could not be considered a factor, because we used so little sedation. Medication for adults took the form of morphine and atropine. Children received either no medication, or small doses of atropine (1/500 or 1/250 gr.), according to age. Atropine has its proponents, who welcome the lessened secretion in the presence of ether which it causes; and opponents, who fear that although the secretion is lessened, its thickening is a source of danger. It is our experience that atropine is valuable in small dosage, especially when the anesthetic is ether, because it may help to prevent bronchial obstruction and atelectasis. We cannot believe that the viscid secretion it may possibly bring about is as likely to obstruct as is the greatly increased secretion produced by ether without atropine. We do know that in pneumonic processes, where there is considerable secretion present, small doses of atropine are distinctly helpful. The preponderance of opinion does not favor any medication that abolishes laryngeal reflexes.

In considering intrinsic factors, we realized how important was the preoperative condition of the patient. Tonsillectomy is an operation of choice, and is rarely to be considered an emergency. We are convinced that the most potent cause for pulmonary complication is infection already present in the upper or lower respiratory tract before operation. With lowered resistance, and the reactivation of an already present infection to virulence, the complication may be produced. Only re-

cently we saw a patient who was prepared for tonsillectomy, which was not performed because of infection present in the lower respiratory tract. This patient developed a lung abscess, which would have been laid at the door of the tonsillectomy. We are convinced that the major source of lung abscess following tonsillectomy is the preoperative condition of the respiratory tract.

As to the type of anesthesia, lung abscess occurs when local anesthesia is used, but quite infrequently. Most clinics record a minimal percentage. It is true that abolition of the throat and laryngeal reflexes allows for the easy entrance of blood and septic material from the throat and tonsils into the trachea, but it does not follow that this in itself is sufficient to cause a pulmonary complication. Myerson[11] examined bronchoscopically 100 cases in which tonsillectomy was done under light general anesthesia, and found blood present in 76, even with the cough reflex present. He found that foreign material was evacuated from the lungs in twelve minutes, even under general anesthesia, in the absence of the cough reflex. The laryngeal reflex does not ensure the presence of cough. Patients will cough only when there is increased irritability in the respiratory tract, under light anesthesia. Iglauer[12] found blood in the trachea in 40 per cent of his patients under general anesthesia, and in 38 per cent under local. It is the prolonged retention of aspirated blood and septic material from the throat that helps to produce abscess.

Although Le Play[13] in 1905, Grossard and Kaufmann[14] and others, began to recognize post-tonsillectomy lung abscess as a distinct entity, it was Richardson[15] who in 1913 first called serious attention to this condition. At the outset he favored embolism; later he became entirely convinced that aspiration was the most important factor. In 1916 Manges[16] reported 9 cases in a single year; he thought they were all aspiratory in origin and blamed the upright position. Yankauer[17] thought that the gaping veins in the tonsillar fossas easily became thrombosed and caused septic infarcts in the lungs. From then on there were many reports from all over the country, until Moore[5] in 1922 called attention to the occurrence of only 202 cases in his series of 450,000 tonsillectomies, a ratio of 1:2200. In all the reports local anesthesia held a very minor place, and general anesthesia was the background in an overwhelming percentage. The fact that most of these abscesses are related to the bronchial tubes rather than to the periphery of the lung distinctly favors aspiration as a cause. Furthermore, the fact that these abscesses occur most frequently in the right lung and in the lower lobes likewise favors aspiration, even though those who favor embolism point to a similar distribution in cases of proved emboli. Bronchial obstruction is so frequent an occurrence that it is difficult to believe that aspiration is not the most natural mechanism of this condition. The work of Coryllos and Birnbaum[18] on pulmonary atelectasis and the convincing evidence of Harkavy[19] are most potent arguments in favor of aspiration, and atelectasis with retained septic material, as the background.

On the other hand, the experimental work of Fetterolf and Fox,[20] Cutler and Hunt,[21] Cutler and Schlueter[22] and others strongly favors embolism. Much experimental work has been done, being designed to reproduce lung abscesses in animals by aspiration and by embolism. Both methods have eventually proved successful. Much doubt has been cast on such experimentation because it has been impossible to be sure that its results were equally applicable to man. The differences in the animal tissues including the pulmonary circulation, together with the normally horizontal position of the trachea in animals as contrasted with the vertical position in man, throw doubt on the validity of these experiments. Moreover, chronic lung abscess has not been produced in animals, because of the tendency of the experimental abscesses to spontaneous healing.

While there is no doubt that lung abscesses occur as a result of aspiration and also by embolism, clinically the preponderance of opinion favors the aspiratory mechanism as the cause in the greater number of cases.

FOREIGN BODIES IN THE BRONCHIAL TUBES

It is striking that in the many foreign-body cases that come to our clinic each year there are not more resultant lung abscesses. This is due to a number of causes. Primarily, physicians and the community at large are more awake than formerly to the possibilities of danger from foreign bodies in the air passages. Likewise, clinics are far better equipped to handle these cases, so that early removal of the foreign body is commoner. Metallic or hard non-opaque bodies do not tend to cause abscess, and in the absence of degenerative substances or infection most of these cases clear up on removal of the foreign body before there is any chance that obstructive infection will occur. It is the vegetable or animal matter tending to cause inflammatory reaction with infection that is most prone to cause abscess. These cases also, when seen early, are likely to recover with adequate drainage as soon as the foreign substance is removed. In our clinic we have had to deal with 188 cases with foreign bodies during this period; only 1 was productive of a lung abscess. In that case, delay in coming to the clinic for

removal of an inhaled veal bone, with consequent infection, was the causal factor.

FACTORS IN CASES FOLLOWING ABDOMINAL OPERATION

In these cases the important factors are the condition of the patient, both before and during the operation; the anesthesia — predisposition to formation of much mucus and atelectasis; and the operative position — interference with lung ventilation, stagnation of secretions, and predisposition to atelectasis; the length of operation; the infective source; and the postoperative period.

These factors are essentially the same as those which are operative in abscesses which follow medical conditions or operations on the upper respiratory tract. The condition of the patient is apparently always the most potent factor. The anesthesia and operation are both of longer duration; the dependent operative position maintained so long, causing interference with lung ventilation and consequent stagnation of secretions, adds to the possibility of atelectasis. Although infarction and embolism are stressed by many, there is much evidence that atelectasis and bronchial obstruction constitute the most frequent cause. Examination of our histories showed 1 possible infarct in a case of tonsillectomy, 1 of definite atelectasis following an appendix operation, 1 of infarct following a gall-bladder operation, 1 of infarct following an operation for gastric ulcer, 1 of infarct following a perineal operation, and 5 of infarct or embolus following gynecologic and obstetric operations. It is possible that there were more infarcts or embolic cases, but there was no evidence to prove their presence. It was only in the gynecologic and obstetric cases that infarct and embolism were prominent, as is the common experience. Most of the other cases more nearly approximated what one would expect to find in atelectatic bronchial obstruction. However, in spite of the fact that the distribution of emboli is often the same as in cases definitely aspiratory, it is reasonable to assume that the peripheral portions of the lung would be more frequently affected in infarction and embolism than those near the bronchi. Nevertheless, those cases with a definite infective source, such as a ruptured appendix or an empyema of the gall bladder, are the most likely ones to develop an infarction or embolus, even though we have been unable to find proof of this in individual cases.

SUMMARY

This investigation represents an analysis of all proved cases of lung abscess occurring at the Boston City Hospital in the years 1926 to 1935, inclusive. There were 196 cases of lung abscess following medical conditions, 4 cases following trauma and 76 cases following postoperative conditions, a total of 276 cases.

Although it has become the general belief that postoperative complications are predominating factors, this was not so in our cases. We believe that a careful investigation in other large clinics would produce results similar to ours. Tonsillectomy is a prominent cause in a large number of cases, largely because it is more frequently performed than any other operation. However, when compared to the total number of operations performed it represents a smaller percentage than is generally realized, and compares favorably not only with the incidence following pneumonia, but also with that following other postoperative conditions.

It is also evident that nearly all the cases of post-tonsillectomy lung abscess occur in adult patients, and that they require more careful selection and preoperative care and better postoperative attention than heretofore recognized.

We believe that a more careful examination before all operative procedures and more careful postoperative observation will considerably lessen the incidence of postoperative lung abscess.

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LUNG ABSCESS AND ITS RELATION TO SURGERY OF THE UPPER RESPIRATORY TRACT

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THERE are two modes of approach to the statistical study of lung abscess. The internist sees enough of this disease in the course of years to be profoundly impressed, not only by its seriousness but also by the fact that in many instances it follows some surgical procedure in the region of the mouth, nose or throat, and particularly tonsillectomy. The otolaryngologist, on the other hand, sees in this attitude a challenging implication with which he takes issue.

The otolaryngologist rarely if ever sees lung abscess as a complication in his operative cases. Furthermore, studies of large series of cases show an average incidence of 1 case of lung abscess in 2000 or 3000 cases of tonsillectomy.[1, 2] Some writers, especially in England, have even found the incidence greater after general operations than after surgery on the respiratory tract. At this point, however, it should be stressed that the important factor is not the ratio of this complication to a given number of operations, of whatever kind, but the very serious fact that in any substantial group of lung-abscess cases, a large proportion occur after operation on the upper respiratory tract, and particularly after tonsillectomy.

The observation of Norris and Landis[3] is pertinent to this matter. They say:

> Although the experience of any one operator, or even that of a large clinic, is limited, the number of cases of abscess following tonsillectomy is in the aggregate quite large. Statements of individual operators that they have never met with this sequel are of doubtful value, and this is especially true of large clinics where the patients are easily lost trace of. It is now known that many of these abscess cases never come to the knowledge of the operator. Some of them are recognized by another physician as abscesses only after months have elapsed.

In 1925, Lord[4] reported 227 cases of lung abscess observed at the Massachusetts General Hospital between 1909 and 1924. Of these, 96 (42 per cent) followed some operative procedure; 78 (34 per cent) followed some operation on the upper respiratory tract, of which 21 (9 per cent) occurred after the extraction of teeth and 49 (22 per cent) after tonsillectomy, and the remaining 8 followed operation elsewhere. Aspiration of foreign bodies was responsible for 8 cases. There were 28 cases (12 per cent) in this series which were ascribed to pneumonia. One case followed esophagoscopy, and in the rest the cause was not determined.

In 1934, King and Lord[5] reported a second series of 210 cases of lung abscess, observed at the Massachusetts General Hospital between 1924 and 1932. While in Lord's earlier series, just mentioned, approximately 1 of every 3 cases could be traced to operations in or near the upper respiratory tract, the proportion in the second series rose to about 1 of every 2 cases (117 cases, or 56 per cent). The operative procedures included the removal of tonsils and adenoids in 81 cases (39 per cent), the extraction of teeth in 26 (12 per cent) and an upper respiratory operation in 10 (4.8 per cent); 19 (9.0 per cent) of the cases were due to other operations under a general anesthetic, making a total of 136 (65 per cent) referable to previous surgical procedures. The remaining cases were less clear; in 48 (23 per cent) the onset was insidious and the cause was undetermined; 17 (8 per cent) suggested pneumonia as a cause; 5 (2 per cent) followed an upper respiratory infection.

In the records of the Beth Israel Hospital there is to date a series of 66 cases of lung abscess. Of these, 34 (52 per cent) came after some surgical procedure; 31 (47 per cent) followed operations on the upper respiratory tract, of which 4 (5 per cent) were the extraction of teeth, and 26 (39 per cent) tonsillectomy. One case followed aspiration of a foreign body, 20 (30 per cent) pneumonia, and the rest miscellaneous conditions, such as the breaking down of carcinoma of the lung, bronchiectasis or some undetermined antecedent condition.

One significant conclusion must be drawn from these figures: surgery of the upper respiratory tract and pneumonia are responsible for most of the cases of lung abscess. The latter condition must be considered in the general problem of pneumonia control. Its incidence following operations on the upper respiratory tract, on the other hand, challenges the serious co-operation of the general practitioner, internist, otolaryngologist, oral surgeon and anesthetist. In fairness it must be admitted that the well-trained otolaryngologist rarely meets this complication. Nevertheless, if from a fifth to a third of all cases of lung abscess follow tonsillectomy, the profession, led by the otolaryngologist, must insist on a better general technic for this and kindred operations.

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From the point of view of the prevention of lung abscess, several propositions stand out:

1. Tonsillectomy and other operations on the nose and throat are major surgical procedures.
2. Such operations should be performed in a hospital, and the cases should be carefully followed.
3. Local anesthesia is preferable to general, but more important than the type of anesthesia is the qualification of the anesthetist.
4. It goes without saying that the surgeon should be well qualified, and also that he should have his patient physically fit before the operation.
5. It is well established that the danger of lung abscess after tonsillectomy is much greater in adults than it is in children.
6. The extraction of teeth should be done in several sessions, obviating general anesthesia and too large a wound, and also minimizing the danger of foreign-body aspiration.
7. Finally, tonsillectomy should not be advised, particularly in adults, without clear and weighty indications. The surgeon and family physician must be sure that the operation is absolutely necessary.

Table 1. *Antecedent Conditions to Lung Abscess.*

Authority	No. of Cases	Following Pneumonia and Acute Respiratory Infections		Operations of All Kinds		Following Surgery: Operations on Upper Respiratory Tract		Tonsillectomy		Extraction of Teeth		Aspiration of Foreign Bodies		Miscellaneous	
		No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
Lord, 1909-1924	227	28	12.3	96	42.3	78	34.4	49	21.5	21	9.2	8	3.5	113	49.7
King and Lord, 1924-1932	210	22	10.5	136	64.7	117	55.7	81	38.6	26	12.4	—	—	48	22.8
Beth Israel Hospital, 1928-1937	66	20	30.3	34	51.5	31	47.0	26	39.4	4	6.0	1	1.5	11	16.6

This discussion merely re-emphasizes what has been taught over and over again. Reiteration, however, will have been justified if it leads in any measure to the prevention of a disease which is so often crippling and even fatal.

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THE GENERAL PRACTITIONER AND THORACIC SURGERY

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PHILADELPHIA

THORACIC surgery is developing so rapidly at the present time that almost monthly one hears of some new development making possible the control or cure of diseases previously considered unapproachable or inoperable. This branch of surgery is so recent that many physicians have not had the time or the opportunity to acquaint themselves with its accomplishments or possibilities.

It seems a very short time ago that 80 per cent of patients with advanced pulmonary tuberculosis died and 20 per cent recovered. Since the intelligent applicaton of collapse therapy these percentages have been reversed. It is still difficult to convince physicians that surgical drainage of lung abscess will cure many of their patients if done early enough to avoid the bronchiectasis which accompanies long-standing suppuration. There is work now afoot which, when evaluated, may prove that drainage within the first two or three weeks applied to all lung abscesses will greatly reduce the morbidity and the mortality of this serious disease. This branch of surgery has also made possible the cure of bronchiectasis, and is a possible modicum of control of carcinoma of the lung, though this is not yet proved. The demonstration of a few cases may illustrate some of the possibilities.

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Presented at the annual meeting of the Vermont State Medical Society, St. Johnsbury, October 15, 1937.

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DIAGNOSTIC AIDS

Since diagnosis is so essential to proper treatment, a few words on some of its aids will be appropriate. They are as follows:

1. X-ray. Extremely important and helpful.
2. Bronchoscopy. Extremely important.
3. Bronchoscopic biopsy when tumor is visible.
4. Sputum analysis. Important.

5. Pneumothorax. Often helpful.
6. Thoracentesis when fluid is present, with histologic study of sediment.
7. Thoracoscopy. Limited usefulness.
8. Needle-punch biopsy. Often helpful.

Except for a careful history-taking and a thorough physical examination, x-ray is doubtless the most helpful diagnostic procedure. Unfortunately there are frequent complications which decrease its value. One of these is the presence of effusion in the pleura, which gives a homogeneous density without diagnostic detail.

A boy of eight (Case 1; Fig. 1) had persistent vague symptomatology and a pleural effusion preventing x-ray diagnosis. Removal of approximately 1600 cc. of fluid with instillation of air enabled the x-ray to reveal a large mediastinal tumor, which was proved at postmortem examination to be lymphosarcoma.

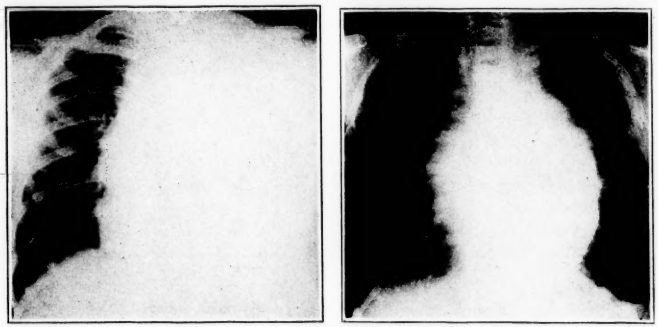


Figure 1. *Case 1. Before and after removal of fluid and reinstillation of air, demonstrating large mediastinal tumor.*

A man of fifty-one (Case 2) complained of cough and malaise for 1 year. Four months previously he had developed dyspnea, and effusion was found. The fluid obscured any underlying lesion, but when it was removed and replaced by air, a tumor of the left lower lobe was revealed, with some irregularity of the pleural surface indicating metastasis.

For either diagnosis or therapy large amounts of fluid can be removed at one sitting if one will equalize the intrathoracic pressure by injecting, almost simultaneously, equivalent amounts of air so that the lung is not rapidly expanded or the mediastinum rapidly shifted. The amount of fluid removed can exceed the amount of air reinjected by about 1000 cc. in the average adult. We have done this on many occasions and have so far seen no ill-effects, other than those which may follow the single insertion of a needle.

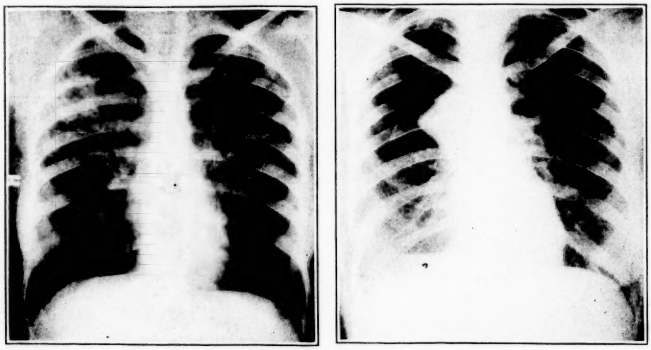
Bronchoscopy in expert hands is quite harmless and is extremely helpful both in diagnosis and in therapy. In another patient (Case 3), however, diagnostic tissue from the bronchus was unobtainable until a second attempt had been made. Because of this, two other valuable methods of diagnosis were applied: lipiodol was instilled for a bronchogram, which demonstrated the lesion to be fairly high in the lower lobe bronchus; and a thoracoscope was inserted through a trocar between the ribs for examination and biopsy of the irregular pleural surface. Histologic study of the tissue proved the tumor to be an adenocarcinoma.

Thoracoscopy, although limited in its usefulness, is particularly helpful in selected cases. A patient (Case 4) now in the hospital, entered complaining of cough and dyspnea and was found to have a large pleural effusion; when this was evacuated and replaced by air, there was revealed a large carcinomatous infiltration of the anterior wall and diaphragm, originating from a recurrent carcinoma of the right breast removed eight years before. This infiltration also was biopsied through the thoracoscope, since there was no endobronchial tumor to be obtained through the bronchoscope.

TUBERCULOSIS

Unless one can produce closure of cavities in tuberculosis, cure cannot be obtained. Although in many patients this result is achieved by sanatorium care, many others are not so fortunate. Sanatorium care alone is usually quite prolonged and expensive. The principle underlying the surgical attack on tuberculosis is closure of the cavity by the various methods of collapse therapy. Pneumothorax is the simplest and most popular method of producing such collapse, and there are very few

who prefer other methods when pneumothorax can be performed. The following patient (Case 5; Fig. 2) obtained excellent collapse and permanent cure even after re-expansion. Pneumothorax was begun four months after the onset of a rather acute illness, and was continued for seventeen months. The lung was then allowed to re-expand, and there was no evidence of continuing disease. The patient has been clinically well for about three years. How long such pneumothorax should be continued and whether it should ever be abandoned are controversial subjects. Certainly it should not be terminated in less than three years, and present opinion seems to favor continuing it as long as possible. Unfortunately, in many cases the pleural space is gradually obliterated by adhesions in spite of all efforts to maintain the pneumothorax.



A B

Figure 2. *Case 5. A. Before pneumothorax, multiple cavitation of the right upper lobe. B. Five months later, selective pneumothorax, with complete collapse of right upper lobe and only partial collapse of right lower and middle lobes.*

Frequently pneumothorax can be only partially obtained because of adhesions from the chest wall to the lung around the cavity, which hold it open. If the cavity shows fairly thin walls and the adhesions are long and tenuous, they can be cut by thoracoscopic technic without opening the chest. The result of such intrapleural pneumolysis is illustrated by Case 6, that of a young woman who had had pneumothorax for nine months, with continued productive sputum and only moderate general improvement. Following severance of the adhesions there was rapid improvement, the sputum became negative within two weeks, and the cavity closed. Seven months later she was clinically well, showed no cavity and was going about her usual household duties, returning every two weeks for pneumothorax refills.

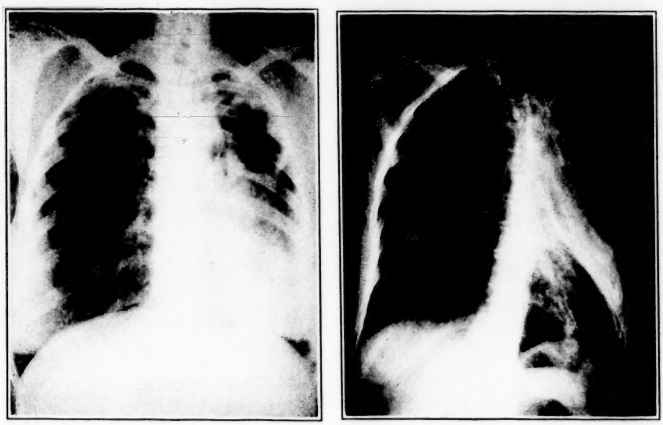
If several adhesions are present in the region of the cavity, pneumothorax is often inadequate or unobtainable. Under such circumstances, paralysis of the phrenic nerve, which allows the diaphragm to ascend into the thorax, relieves the tension upon the lung, and may produce sufficient relaxation to allow closure of the cavity. Unfortunately this method is frequently ineffective, often produces considerable dyspnea, and interferes with effective expectoration. For these reasons thoracoplasty is usually preferred. If phrenic surgery is attempted, it should be done as a temporary measure, and should consist in crushing the phrenic nerve, which will paralyze it for from three to nine months. If this procedure is ineffective or harmful, the paralysis is temporary; if, on the other hand, it is effective and too brief, the nerve can be removed at a subsequent operation in order to induce permanent phrenic paralysis. The patient in Case 7 was ill for one year and was unsuccessfully treated with pneumothorax. Her sputum was negative within two months after phrenic surgery, and she has been clinically well for four years.

As a rule, when pneumothorax is ineffectual, thoracoplasty, either partial or complete, is the procedure of choice; enough of each rib overlying the involved area should be removed to produce collapse of the chest wall sufficient to close completely the underlying cavities. This patient (Case 8; Fig. 3) obtained such a result and a clinical cure six weeks after thoracoplasty. This was done in two stages, six ribs being removed and the function of the uninvolved lower portion being preserved.

EMPYEMA

The increasing knowledge of intrathoracic physiology has contributed also to the treatment of empyema. By the older methods of wide and adequate drainage, as many as 30 per cent of simple empyemas may result in death when such wide-open drainage is applied early to a massive effusion, before stiffening of the mediastinum or adhesions of portions of the lung to the chest wall have occurred. Open drainage at this time destroys the negative pressure by which the lung is kept expanded and by which the two sides of the thorax are equalized in pressure. The result is sudden and complete collapse of the affected side, with attendant dyspnea and anoxemia and movement of the mediastinum with each respiratory effort. This mediastinal flutter causes severe embarrassment to the heart, and we have seen such patients, recovering from their infection, die of the failure of an otherwise normal heart. If, on the other hand, a closed method of drainage is utilized, we maintain equilibrium between the two pleural spaces and a constantly expanding lung. This obviates mechanical difficulties arising immediately after operation, and the lung is pulled out to the chest wall as the pus is evacuated. It adheres shortly after contacting the parietal pleura, thus obliterating the pleural space, and prevents the formation of thick-walled chronic empyema cavities, which necessitate multiple deforming operations such as extensive thoracoplasties. Such large chronic cavities are practically unknown under this method. In addition, the illness is greatly shortened. It is thus possible to use this method on both sides at the same time, since the respiratory mechanism is continued at an increasingly excellent level. This patient (Case 9; Fig. 4) was so treated for bilateral empyema following bilateral pneumonia. He made an uneventful recovery in spite of the drainage on the two sides within five days of each other, and was discharged five weeks after the second drainage.

In a fair percentage of cases following such closed drainage, a pocket of loculated empyema results, necessitating a second operation. With such loculated cavities, the lung being adherent to the chest wall about the pocket, open drainage is safe and preferable. If the empyema when first seen is already loculated and fairly small, time is saved by immediate rib resection and open drainage, with the removal of the fibrin clot which is usually present. This was so in Case 10, the patient being discharged three weeks after drainage had been instituted. We consider aspiration valueless except for diagnostic purposes and the misconception, so prevalent in some communities, that repeated aspiration is a conservative and safe treatment of empyema has caused many deaths, countless complications or at best a great increase in morbidity.



A *B*

Figure 3. *Case 8. A. Large cavity, left upper lobe and ineffectual pneumothorax. B. Pneumothorax abandoned and the cavity closed by two-stage thoracoplasty.*

LUNG ABSCESS

Although the percentage of lung abscesses among the patients of any one doctor or hospital is small, the lesion is important because of its serious character and the high probability that it will become chronic and extremely debilitating, if not fatal.

In spite of experimental evidence to the contrary, the majority of lung abscesses appear to be due to various forms of aspiration of infectious or irritating material. Consider the patient who, under general anesthesia, inspires vomitus, blood, pus, tissue or some other foreign body while the protective cough reflex is abolished. Other sources of unconsciousness, such as drunkenness, trauma, drugs and even, rarely, natural sleep may permit aspiration similar to that under anesthesia. Following any such a period of unconsciousness we may have the onset of cough, fever and pain in the chest, with physical signs of consolidation, followed in two or three weeks by the profuse production of foul-smelling sputum as the abscess breaks into a bronchus.

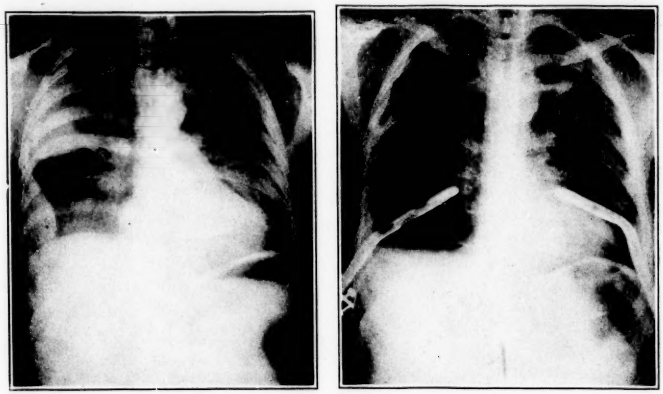


Figure 4. *Case 9. Before and three weeks after simultaneous drainage of bilateral empyema.*

Other etiologic factors fairly commonly observed are respiratory infection, such as bronchitis or pneumonia, which fails to clear up; infected emboli from other sources, which occasionally lodge in the vessels of the lung and start an abscess about them; and, rarely, trauma such as that caused by a gunshot or stab wound.

In any case, the great majority of patients presenting this picture fall into three main classes. (1) When evacuation into the bronchus occurs, it is effected by means of a large opening which furnishes adequate drainage, the patient has a good resistance to the infecting organisms, and there is a complete liquefaction of the destroyed tissue. These conditions are conducive to spontaneous recovery, which occurs in approximately one third of all cases. In Case 11, the patient showed definite improvement in two weeks and a complete cure in six weeks from the time the abscess was discovered. If there is no definite improvement in two months from the time of discovery, further delay in obtaining drainage by surgery seriously jeopardizes the patient's chances for recovery; for spontaneous cure is almost never obtained, and a constant absorption of toxic material causes progressive debilitation, while the lesion frequently spreads to adjacent parts of the lung because of recurrent temporary blockage of the drainage aperture. (2) Bronchoscopic aspiration and postural drainage, combined with blood transfusions and other supportive measures, may increase the percentage of nonsurgical cures from 30 to 40 or 45. (3) Of the remaining 60 or 55 per cent of these chronic and long-standing abscesses, adequate surgical drainage may cure an additional 20 or 25 per cent. An example of the excellent response to drainage is furnished by Case 12, in which surgery was not postponed too long. The patient was completely well in ten weeks, although he had been ill for five months before drainage. A film taken one year later showed no evidence of recurrence, and the

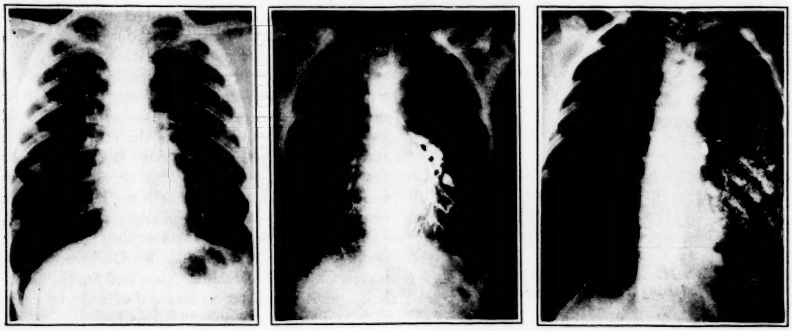
patient has continued his usual activities in excellent health.

Almost always if such drainage is delayed for several months, and occasionally even when it is instituted fairly early, there has been so much destruction of lung tissue and so much bronchiectasis that the lobe or even the whole lung is destroyed, and lobectomy or pneumonectomy is the only possible means of cure.

BRONCHIECTASIS

At any age, chronic cough, with profuse expectoration and physical signs of moisture in certain parts of the bronchial tree, with or without hemoptysis, fever, toxemia or signs of complications, are frequently caused by bronchiectasis or tuberculosis. Such patients often reveal no physical signs of consolidation, and even x-ray, unaided, may not suffice to show the true condition. Without the assistance of opaque material, such as lipiodol, to delineate the size and shape of the bronchi, the diagnosis will often be missed. In Case 13 (Fig. 5), one sees few abnormalities in the first film, and no more were shown until the lipiodol was instilled. This is mainly because the area which might have shown induration is obscured by the cardiac silhouette. The etiologic factor in this case was a tonsillectomy done four years previously, but many such cases are congenital, and show no symptoms until some respiratory infection later in life leaves the patient with typical bronchiectatic symptoms. Many other cases develop from continued respiratory infection, such as a chronic sinusitis or even tuberculosis. Any condition producing prolonged lavage of the bronchi by purulent material may induce bronchiectasis.

Although we have never seen a patient with this disease permanently cured by any means other than surgery, occasionally for fairly long periods such cases may be made symptom-free by bronchoscopy, postural drainage, supportive measures, vaccines and so forth. Nearly always, however, the next respiratory infection brings a recurrence of the symptoms, often in a more severe form. The risk of lobectomy is becoming less, and from 50 per cent in 1916 the mortality has dropped to approximately 15 per cent, as shown by a recent analysis of 212 cases in the hands of many different surgeons. In Case 12, the left lower lobe was removed under local anesthesia, and the patient was discharged completely well six weeks later.



A *B* *C*

Figure 5. *Case 13. A. Some increase in peribronchial density, but insufficient to diagnose bronchiectasis. B. Explanation by lipiodol instillation. C. Eleven weeks after left lower lobectomy under local anesthesia. Lipiodol reveals a normal bronchial outline in an emphysematous left upper lobe and the stump of the left lower lobe bronchus filled with lipiodol.*

BRONCHIOGENIC CARCINOMA

The supposedly rare condition of bronchiogenic carcinoma of the lung is found upon study of both postmortem and hospital admission statistics to occur in approximately 10 per cent of all cases of carcinoma. It therefore behooves us to keep it constantly in mind, especially since the symptoms

of the early stage are extremely vague. The only constant symptom is that of mild cough, which is usually disregarded by the patient and even by the physician. Thus it appears essential that wherever a persistent cough, especially in a person above thirty-five, does not respond to active treatment, the case be investigated to the limit of our diagnostic ability before carcinoma is excluded.

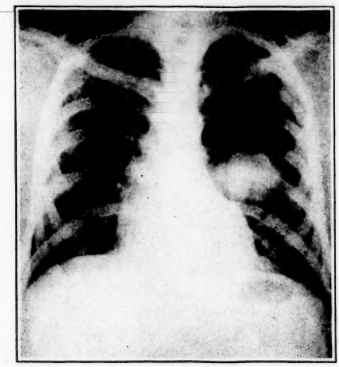


Figure 6. *Case 14. A medium-sized tumor of left upper lobe revealed following a diagnostic pneumothorax.*

Hemoptysis from ulceration of the tumor also may occur fairly early. However, atelectasis, pain in the chest and pleural effusion, any one of which may cause the patient to seek aid, usually indicate an advanced involvement, and such cases often prove inoperable. Frequently it is impossible to know the operability of such a lesion without thoracic exploration. We have explored 9 such cases and found only 1 operable. This patient (Case 14; Fig. 6) was only twenty-seven, and was first thought by his physician to have a bronchiogenic spread of tuberculosis from a peribronchial lymph node. Thus considerable time was wasted, and the very cellular cancer grew rapidly until lobectomy was performed, seven months after the first complaint of cough and hemoptysis. The patient made an uneventful recovery from the lobectomy, but because of our mistaken conservatism in removing only the involved lobe and not the entire lung and mediastinal lymph nodes, he returned in four months with recurrence in the lower lobe at the hilus of the removed upper lobe. A second attempt was made to complete the pneumonectomy and remove the tumor, but it was so soft, rapidly growing and extensive that the operation had to be abandoned. He was treated by heavy doses of x-ray therapy without effect, and the patient died eighteen months later.

In spite of the marvelous results obtained by x-ray in many carcinomatous lesions, no proved case of bronchiogenic carcinoma has ever been cured by this method, and as a rule the effect upon the tumor is so slight that it does not compensate for the sickness which so frequently accompanies heavy dosage. The only benefit we have seen has been that of the abolition or decrease of pain.

Although it is too early to evaluate the results from surgery, several articles by Graham, Sauerbruch, Churchill, Edwards, Allen and Smith, Overholt and Rienhoff report upward of a dozen cases alive and apparently well for more than six months. Two of these have been observed for three years after operation, 3 for two years and 2 for one year. Thus one can visualize the probability that earlier diagnosis made by alert physicians, who will urge radical surgery upon such patients as their only hope of cure, may eventuate in a measure of control of bronchiogenic carcinoma similar to that of cancer in other parts of the body.

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THE CLASSIFICATION AND TREATMENT OF ARTHRITIS

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THE two most important factors underlying the confused state of mind of the medical profession in regard to arthritis are: first, the lack of a universally understandable etiologic classification, and secondly, a failure to understand the pathologic process which goes on in the joint tissues in response to irritation. If a classification based on the pathologic processes in the joints gave any adequate idea of the processes themselves, it would serve admirably and the confused state of mind would cease to exist. The fact that arthritis is so poorly understood, even at the present time, is convincing evidence that pathologic classifications have failed. The etiologic classification here presented has been used in instructing nurses and medical students concerning arthritis, with a resultant grasp of the subject which is most encouraging, considering the difficulty experienced with the prevailing classifications.

This classification is based on the theory that there are three basic etiologic factors in arthritis: bacteria, toxins and trauma. It is well known that bacteria may exert their deleterious effects upon tissues in two ways: first, by invasion of the joint tissues, and secondly, by the poisonous effects of the products of bacterial metabolism. The latter does not require the presence of the bacteria in the joint tissues, and the source of production of the poisons — the focus of infection — may be in a distant part of the body. If the bacteria are present in the joint tissues, both bacteria and the products of their metabolism operate, but the effects of the former are so much more severe than those of the latter that the latter are submerged.

The very fact that no distinction is made between direct invasion and poisoning by bacterial products constitutes a predominant source of confusion in the understanding of arthritis, and the principal bar to proper treatment. The effects cannot be the same. The presence of bacteria in the joint tissues calls forth the white blood cells, principally the polymorphonuclears, to combat the invasion. The proteolytic enzymes elaborated by these cells bring about the liquefaction of tissues, which are destroyed in the inflammatory process. The destructive action in infection is in inverse proportion to the intimacy of blood supply to the tissues and in direct proportion to its lack. The articular cartilage, having a very feeble blood supply, has little if any defense against the destructive action of the proteolytic enzymes and is an easy prey. Bacterial poisons, on the other hand, call out very little polymorphonuclear reaction, and the tendency to destructive liquefaction of the cartilage is consequently less.

Thus, destructive action on the cartilage — the essential joint tissue — and its inability to regenerate are fatal to the joint as a moving mechanism. Between the extremes of virulent bacterial invasion and bacterial toxic damage are gradations of joint damage depending upon the polymorphonuclear reaction developed. In the classification here presented, "infectious" has been made one of the main headings; this group has been subdivided into "bacterial" and "toxic," so as to recognize the production of joint damage by direct bacterial reaction and by the toxic action of bacterial products.

In bacterial arthritis, a focus of infection in another part of the body is of minor importance, since the establishment of the bacteria in the joint tissues constitutes an autonomous focus. Knowledge that the bacteria have invaded the joint is therefore of major importance, as is recognition of the particular organism and its characteristics. In toxic arthritis with an infectious basis — the focal-infection type — it is necessary to locate the focus of infection in order to recognize the organism. Elimination of the focus will contribute much to improving the condition, but the degree of improvement and the period of time necessary for it to take place depend upon how long changes in the joints had existed before the focus was removed, and the care of the joints during the active infection. Furthermore, discovery of a focus of infection does not necessarily mean that it is the sole or even partial cause of the arthritis.

It often happens that the focus is so located that it cannot be eliminated, and to minimize the effects of this infection will often tax one's ingenuity. The question may also be raised in so-called focal infection whether foci are the causes of the joint affection, or merely manifestations of a lowered resistance to infection. Even when improvement in the joints soon follows elimination of a focus, the latter is not necessarily the cause of the arthritis, since it may well be considered an overload on the body economy, the removal of which placed the body mechanism in a better position to combat the joint condition.

All arthritis is not caused by infection, and all toxins are not the product of bacterial action. In order to include those causes which do not arise

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from infections, and to devaluate the overwhelming importance which has been ascribed to infection since the promulgation of the theory of focal infection, the other main heading in this classification has been designated as "noninfectious." The principal subdivision under this heading is "toxic." This term is a logical and desirable one. Under it can be placed the many diverse and seemingly unrelated causes of arthritis which are gaining adherents with the development of our knowledge concerning the abnormal actions of various glands in the body, and with the discovery of the effects on the body of poisons originating outside it. Since this classification, if it finds favor, must serve in the investigation of arthritis as well as act as a convenient clinical guide, the term "toxic" requires further subdivision, according to whether the toxins arise within or outside the body. The term "endogenous" includes those conditions other than infection which within the body give rise to toxic products. The terms "allergic," "metabolic," "climacteric" and many others which have no logical place in any existing classification may be readily placed in this division as "glandular dysfunction" or "intestinal dysfunction," without doing violence to our classification.

"Exogenous" poisons include those arising outside the body but taken into it accidentally — through environment or industrial exposure, — or intentionally, — as food or medicine, — without knowledge of their poisonous effects on the joints. Arthritis and arthritic symptoms are not always primary, but may be secondary to disturbance of the muscular control of the joints from various causes. If the cause of the disturbance be unknown, the latter would be considered the etiologic factor, and the joint condition is to be classified under the second subheading "noninfectious, traumatic," the subclassifications of which will be discussed later. But if the cause is known to be chronic lead poisoning, for instance, the arthritis is to be classified under "noninfectious, toxic."

Trauma, the third important etiologic factor, finds a logical place under the heading "noninfectious." All joints are not equally subject to trauma, but that trauma is a cause of arthritis needs no demonstration. It is rather the definition of just what constitutes trauma that requires explanation. The joints suffer trauma in their ordinary use. Without any specific trauma, the joints after many years show certain changes which are designated as hypertrophic arthritis. Certain factors tend to bring these changes about at an earlier age than they would ordinarily occur. Just when they are to be considered normal and when pathologic cannot be stated here, but the age of the person affected and the extent of their development determine their abnormality.

Habitual variations from the normal physiologic posture continued over a long period — faulty posture — result in strain of the joint structures, giving rise to changes and symptoms before they would normally be expected. Such trauma is classified as "physiologic." If, in the performance of a particular occupation, one or several joints are brought into use repeatedly many times a day for a long period, the joints so used will show a greater development of abnormal changes than others. This type of trauma is designated as "occupational." If, as the result of a violent injury to the structures about a joint, its normal relations are disturbed, the eventual changes and symptoms are designated as "accidental."

For ordinary clinical use, the classification as outlined is no doubt too detailed. By judicious elimination and consolidation, it can be reduced to the three essential etiologic factors from which we started.

Table 1. *Classification of Arthritis.*

ACUTE OR CHRONIC

- INFECTIOUS
  - Bacterial
    - *Bacillus tuberculosis*
    - *Treponema pallidum*
    - Streptococcus
    - Gonococcus
    - Staphylococcus
    - Etc.
  - Toxic

Source	Organism
Teeth	*Streptococcus hemolyticus*
Tonsils	*Streptococcus viridans*
Sinuses	Staphylococcus
Prostate	Pneumococcus
Gall bladder	Etc.
Etc.	

- NONINFECTIOUS
  - Toxic
    - Endogenous
      - Intestinal dysfunction
      - Glandular dysfunction
    - Exogenous
      - Foods
      - Medicaments
        - Arsenic
        - Lead
        - Radium
      - Biologic Materials
        - Serums
        - Insulin
      - Industrial poisons
- TRAUMATIC
  - Physiologic
  - Occupational
  - Accidental

Simplification of Etiologic Classification

It should not be inferred that any case of arthritis will be confined in its etiology to any one or even two of the factors given. No one with an adequate present-day understanding of arthritis expects to see many cases of a single type. Trauma is frequently a localizing factor in a bacterial arthritis, but the latter is classified as infec-

tious since the symptoms and signs are those of infection and the trauma is incidental. After an infectious lesion has occurred in a joint without obvious damage, the mechanism of the joint is often sufficiently disturbed so that with ordinary use changes will arise which must be classified as traumatic. The presence of toxic factors in the body in conjunction with the abnormal use of a joint results in earlier or more pronounced changes of a traumatic nature, and the condition is to be accordingly classified.

A classification is not a substitute for reasoning; it is a logical and convenient arrangement based upon knowledge of the subject, and as such is presented in Table 1.

* * *

Arthritis, whether it be acute or chronic, is a painful disease. It interferes with, if it does not prevent, normal activity of the body, and it constitutes a threat to joint motion. For these reasons it requires positive and intelligent treatment, which in turn demands a complete understanding of the factors behind the arthritic process.

As stated above, there are three principal foes to normal joint function: bacteria, poisons and trauma. While any one of these may act alone to produce joint lesions, it is more likely that two or even all three will act in a given case. All the possible combinations of these three factors cannot be considered in a short article, and it is doubtful whether even a fair-sized volume could cover the large number of ways in which the three may interact. We shall discuss the treatment of the pure arthritic types, but the practitioner must combine with these conclusions his study of the individual case.

BACTERIAL ARTHRITIS

The indefinite term "infectious arthritis" has come to convey different impressions to different people. The term "bacterial arthritis" is used here to denote any arthritis caused by invasion of the joint structures by bacteria. How is bacterial invasion to be determined? For the time being, only recovery of the bacteria from the joints affected can be considered scientific proof, although clinical judgment may at times determine the matter without resort to identification of the organism. If the organism be the tubercle bacillus, the treatment must be limited to what the patient is willing to accept. At present, permanent control of the tuberculous process is possible only through the destruction of the joint. Retention of varying amounts of joint function with subsidence of the tuberculosis is possible, but the reactivation of the tuberculous process is a constant threat.

Luetic arthritis, like syphilis in any part of the body, is susceptible to general treatment by specific drugs, but the time element here is as important as in any invasion of the joint; that is, procrastination may result in permanent joint damage even though the luetic process is eventually brought under control by antiluetic drugs.

For practical purposes, all the pus-producing organisms can be considered in a group. Invasion of a joint by any one of them may result in an inflammatory reaction, varying from a serous joint effusion to a fulminating purulent one. Destruction of cartilage, the tissue peculiar to the integrity of the joint, is accomplished by the proteolytic enzymes elaborated by pus cells. The number of pus cells present, determining as it does the concentration of enzymes, is an important indication of the degree of danger to the cartilage. Three stages of severity are recognized: serous, seropurulent and purulent types.

In the *serous* type the invading organism calls forth little cellular response and the joint fluid contains a relatively small number of pus cells. There is relatively little danger of marked destruction of the cartilage. Clinically, there is only a slight rise of temperature, and the white-cell count and polymorphonuclear percentage of the blood are only moderately increased. Surgery is seldom necessary. A well-molded plaster cast, and measures aimed at building up quickly the patient's resistance, are usually sufficient to turn the tide. Even in this mild type of arthritis a close watch must be maintained to see that the condition does not develop into either of the more serious types. The temperature trend and the white-cell count and polymorphonuclear percentage must be watched closely.

In the *seropurulent* type, the number of pus cells in the joint effusion is sufficient to give it a thin, purulent character. With such a concentration of cells, one must be prepared for radical surgery, and resort to it without hesitation if the temperature trend is unfavorable and the white-cell count and the polymorphonuclear percentage tend to rise. The seropurulent type does not always require surgical measures, but until one has had considerable experience, the surgical approach is probably the safest for the preservation of the cartilage. It must always be borne in mind that the serous and seropurulent types may be early stages in the highly dangerous purulent type, but that the process may stop before reaching the last-named stage.

The *purulent* type invariably represents a fulminating infection or a late recognition of a joint invasion. The patient's life is endangered, and the damage to the joint cartilage is of secondary consideration. Treatment aimed at saving the patient

serves also to preserve the cartilage. The evacuation of the pus in the joint is essential.

Before undertaking the surgery of infected joints one should study the anatomy of the surrounding structures in order that good drainage, with minimal structural damage and maximal conservation of future function, may be attained. True joint invasions are essentially acute and self-limiting. A distinction should be made between an actively infected joint and one damaged by infection. The end result of infection may be ankylosis by bone or by fibrous tissue, or motion of varying degrees with a mechanically imperfect joint.

TOXIC ARTHRITIS

Arthritis due to poisons may be acute or chronic. The poisons, or toxins, may arise from bacterial invasion in other parts of the body, that is, focal infections; from nonbacterial sources within the body, that is, intestinal and glandular dysfunctions; or from without the body, that is, from foods, drugs, biological preparations or industrial poisons. Arthritis arising from these causes supplies a large group of poorly understood and, so, poorly treated cases. The disease may be primary, directly affecting the joint, or secondary, through disturbance of joint control by the muscles. Because these poisons are general in their action, the affection of the joint may be one manifestation of the toxic effects, or it may be the result of disturbances to other body functions. While the joint must be protected during the activity of the disease process, so as to conserve its function against the time when the disease process will no longer be active, treatment of the disease itself is a problem of internal medicine, and a complicated one.

It would be of small value to describe specific measures of treatment. Each physician has his favorite preparations which seem in his hands to give the most satisfactory results. It is more important to understand the main objectives of treatment, in the order of their importance.

Elimination of Unfavorable Factors

Unfavorable factors may be etiologic elements, and should therefore be eliminated. But even though they are not causative, if they are unfavorable to the body economy they are hindrances to the return of the body to normal, and must be either done away with or minimized as the first step in treatment.

Focal Infection. This is unquestionably an important factor, and one which has received much attention — to the detriment of other factors — since the theory of focal infection was advanced. In a very narrow sense it has been looked upon as the one element which must be found in every case of arthritis. Infection in the teeth and tonsils, because of their easy accessibility, has resulted in their promiscuous removal, often without sound reasons. Infection in the sinuses, the gall bladder, the prostate and the salpingian tubes has received some consideration, but because of the greater difficulty in obliterating them, these organs have failed to become recognized as causes of arthritis. However, these and many other active infections in the body, such as pulmonary tuberculosis and chronic cystitis, may be unfavorable focal infections, treatment of which is necessary if the arthritic process is to be brought under control.

Intestinal Dysfunction. It is of course possible to have a focus of infection in the intestinal tract, such as colitis, which can and does cause arthritis, but even without definite infection, faulty functioning of the intestinal tract may be an unfavorable or even a causative factor in toxic arthritis. The end products of digestion may be thought of as having a normal consistence. If those products are retained in the colon beyond a reasonable time, the consistence increases, indicating that some of the fluid has been absorbed. It does not seem likely that the fluid is absorbed as distilled water, nor that only beneficial products are contained in the solution absorbed. It goes without saying that provision has been made within the body for the removal of deleterious substances absorbed from the intestinal tract, but the detoxicating agency may be operating in a faulty manner. Granting such a train of events, it is not difficult to understand how the intestinal tract may be an unfavorable factor.

Glandular Dysfunction. The glands of internal secretion have profound effects on all body functions. In our present state of knowledge concerning these glands, one can only say that glandular imbalance, in the form either of an excess or a deficiency, may vitally affect the body mechanism, in a way which we as yet understand only imperfectly, in spite of the startling discoveries of the last ten years. When the internal glandular functions and their effects are more clearly understood, we shall be able to treat some types of toxic arthritis more effectively. Today it is known that the exhausted patient with hyperthyroid disease and the lethargic and overweight hypothyroid individual may both suffer from joint difficulty; the basic answer in either case is regulation of the thyroid function. The coincidence of joint difficulty at onset with the natural or artificial production of the menopause is so well recognized that the term "climacteric arthritis" has been used to describe the condition, and ovarian substance has been found beneficial. Some evidence points clear-

ly toward internal glandular factors as included in the etiology of ankylosing arthritis of the spine.

Diet. Faulty nutrition, whether arising from poverty, ignorance of proper diet, faulty metabolism or unwisely attempted control of body weight, is a bar to recovery from arthritis, and in some cases may well be considered a cause of it. All too often, restricted diets as part of the treatment of arthritis aggravate the disease which they are intended to relieve.

Fatigue. Either mental or physical fatigue must not be permitted. It is often one of the symptoms in toxic arthritis, and calls for complete rest until it disappears.

Exogenous Poisons. These may be classified according to their method of ingress, or according to their origin. The latter classification will be used here. We are not so much concerned with acute, spectacular poisoning, which is seldom a factor in the causation of arthritis, as with slow, insidious poisoning recognized by its remote results rather than by its alarming symptoms.

Diets, or special groups of foods, have always been a favorite method for the treatment of arthritis, and never more so than at the present time. It seems as though every possible combination of food had been recommended for the arthritic; on the other hand, all classes and many individual foods have been banned as inimical to his welfare. Foods vary in digestibility, in their bulk or lack of bulk, and in their nutritive value, but they are all foods. The reaction of the body to them varies, and when a food causes a skin eruption, diarrhea, nausea or any other untoward symptom it is a poison to the body. It is called an "allergic substance" and the reaction is called "allergy." The term "allergic arthritis" has found some acceptance but should be shunned because by derivation it explains why something is poisonous instead of naming the cause. The fact that a given food is poisonous to an arthritic patient is sufficient reason to exclude it from his diet. It is not always possible to show a direct connection between the food poison and the arthritic symptoms, nor is this essential, since the poison concerns us as a factor unfavorable to recovery rather than as a cause. Tomatoes, strawberries, certain shellfish and various meats are not tolerated by certain individuals. It therefore devolves upon the physician to check carefully the patient's reaction to the various classes of foods, and to certain foods in particular.

Drugs, which include an ever-widening list of chemical substances and compounds, must have the same close scrutiny as must foods. The patient is not so apt to sense the relation of medicine to dysfunction as he does that of foods. Insulin, not properly a drug but a biologic product, has been known to cause secondary arthritis.

In *industrial poisons* we again have a group of substances in which the relation to illness is not immediately apparent. The individual may come into contact with the substance in industry or through use of a manufactured product containing it. Lead has been found to be a cause of arthritis, as have arsenic and radium.

Restoration of Normal Physiology

While this second objective in the treatment of toxic arthritis must sometimes await removal of the unfavorable factors, in many cases restoration of the various disturbed functions of the body cannot be begun too early. Reliance upon the well-known tendency of the body to resume its normal state after the elimination of disturbing elements has caused many failures in the treatment of chronic diseases. The toxic arthritic is invariably subnormal in weight, and a trend toward normal is one of the best indications of his improvement. "Chronic indigestion" and "dyspepsia" are often but the outward manifestations of a subnormally functioning digestive system. At the beginning of treatment, the patient cannot copy the diet of the normal person, either as to quality or as to quantity. Small, frequent, highly nutritious and easily digestible meals should be the rule, with a gradual return to normal food habits. In patients who are overweight, restriction of carbohydrates is generally recommended.

Elimination through the intestinal organs suffers with the disturbances of the gastric organs and the limitation of general activity. The proper method for such elimination varies with each case. Abdominal massage and colonic irrigation are used in patients with intestinal organs severely deranged, but these must be looked upon as emergency measures, to be dispensed with in favor of milder means at the earliest possible time. The proper selection of cathartics and laxatives is difficult in the face of the glowing promises made for an increasing number of advertised preparations. The physician should familiarize himself with a few good laxatives, and stick to them.

Exercise is extremely important and is, incidentally, a step toward the return to normal bowel habits. The skeletal muscular system of the body, as well as the muscle of many vital internal organs, loses tone and strength in a chronic illness imposing long inactivity. Restoration can come only from careful and systematic training and the physician could profitably imitate the athletic trainer in his efforts toward their end. Appropriate exer-

cises of gradually increasing difficulty for the various muscle groups should be given, always remembering that exercise of a muscle within its fatigue limit strengthens it, and exercise beyond that limit weakens it.

The *skin* is of great importance as an excretory organ in chronic toxic conditions such as arthritis. This function, as well as its heat-regulating one, is invariably disturbed in toxic arthritis. The many small muscles controlling the hair follicles and the sweat glands require development, as well as the larger skeletal muscles. Protection of the skin against sudden and extreme drops in temperature is always necessary in the arthritic, but exposure to the air in a warm atmosphere with transient waves of moderate coolness does much to restore the skin function to normal. Bathing in water of slightly varying temperatures is less practical than bathing in air, and can be recommended only under exceptional conditions. A dry, glazed skin and constant perspiration are but different manifestations of a disturbed skin mechanism.

The *blood*, as in nearly all illnesses, suffers a decrease in the percentage of hemoglobin and in the number of red cells. The functioning of organs giving rise to the white cells is often impaired, but it is difficult to say when this is due to the disease and when to medicaments. When infection is present, the white-cell count is normally increased, as is the percentage of unsegmented polymorphonuclear cells; but because of the disturbed function of the organs giving rise to the white cells, in keeping with the subnormal functioning of the entire body, the cellular response may not follow normal lines. Ordinarily these organs recover without special treatment. Disturbances in the circulation have been studied at some length by Pemberton and his associates. They may be due to the lowering of the heart-muscle tone as well as that in the blood vessels themselves. A lowering of the blood pressure, both systolic and diastolic, would naturally be expected, especially in toxic arthritis of long standing; it returns to normal with increased body activity. It may be well at this point to speak of the importance of the skeletal muscles as accessory organs of circulation. The normal bending of the extremities at the joints, with compression of the veins between the muscles, helps to return blood to the heart. The inactivity of the extremities in arthritis hinders this return of blood, with resultant congestion.

Disturbances of the nervous system are frequently observed. It is not unusual to find the chronic arthritic emotionally unstable or even suffering from "nervous prostration." Psychoses, too, are not uncommon. These are manifestations of the subnormal condition of the nervous system in keeping with the rest of the body, and they respond to measures used to restore the body as a whole to normal.

The *liver* is a most important organ in the treatment of toxic arthritis. Since it is the principal detoxicating organ, any disturbance of its function is bound to have a profound effect on the body's ability to recover from disease. Clinical tests for the determination of its function are not easily available or universally acceptable. It may be presumed that in arthritis the liver in its detoxicative function needs some assistance at intervals. Small doses of calomel followed by a saline cathartic have been found the most useful and dependable aid.

Protection of Joints

In toxic arthritis, which is a systemic disease with joint manifestations, the ultimate permanent joint damage is greatly influenced by the care of the joints during the activity of the disease. Even chronic toxic arthritis may be self-limiting, but recovery from the disease gives small satisfaction if the joint function is so greatly impaired that normal activity is difficult or impossible. *Rest* and *exercise* are the main methods of controlling the health of the joint structures, and heat is a subsidiary aid.

When rest is advisable, and when exercise, are difficult to state in general terms. Each case presents a problem all its own. In general, rest should be used when, owing to inflammatory reaction, any motion of the joint causes pain and muscle spasm. While it cannot be claimed that in rest there is no danger of inducing stiffness, it can be said without fear of contradiction that continued use of the irritated joint will result in changes which will eventually interfere with its motion. Rest during the active inflammatory period, on the other hand, by preventing further tissue irritation and by permitting the inflammatory reaction to reach its end point at the earliest possible time, allows motion to be started earlier and causes less damage to the joint structures.

There are several methods of obtaining rest, the careless application of which does not necessarily bring about the desired result. Rest in bed is presumably the most complete and effective form. Where the entire body requires rest this form is necessary, but not infrequently its attendant conditions make it impossible. In the first place, the bed must be conducive to rest. The author has observed situations in which a mattress which did not permit a change of position prevented the patient from sleeping for several days. The environment must be congenial, and the bed must be neither too hot nor too cold. The inner-spring mattress, so much in vogue at the present time and so widely advertised, is not free from serious

drawbacks. The metal springs apparently siphon off the heat produced by the body, giving the patient a feeling of chilliness which may be serious and certainly is not conducive to restfulness. Bed rest in itself does not insure rest for an injured or diseased spine. Those who have studied the sleeping human being have reported that the subject frequently changes his position. With each change the muscles controlling the spine must change to accommodate to the new position. If the patient remains in one position for several hours, he is sure to feel stiff and uncomfortable. In either case, rest is not obtained. If the spine itself requires rest, a plaster-of-Paris shell made to conform to the natural curves of the body is most effectual. Rest for the various muscles is ordinarily obtained by frequent changes of position; in the shells, however, the muscles are not strained and change of position is not essential.

To obtain rest for a part of the body or a single joint, properly made casts, splints or braces are used. The good which these devices can accomplish is limited by the intelligence and skill behind their application. Too often this step is delegated to inexperienced house officers while the designing of braces and corsets is left to bracemakers and corsetières, who, however competent in their own fields, cannot be presumed to understand the surgical conditions for which their apparatus is to be applied. The fitting of appliances such as braces and corsets should be closely supervised by the physician, and if he is not competent he should call in a consultant who is.

Exercise may be classified as general or local and as active or passive. Here is another field in which the average physician is poorly equipped. His advice is usually to "take some exercise," but he does not lay down the particulars of the exercise, and experience shows that the patient is not a good judge of the type or amount of exercise, any more than he is competent to prescribe the dosage of morphine or digitalis. The judicious combination of exercises of graduated degrees of difficulty within the fatigue limits of the muscles brings the best results.

Motion may be relaxed, passive or active. Relaxed motion is that type in which the muscles of the part itself are inactive but the action of gravity is used to give motion to the part. Passive motion is that type in which a force other than that of the patient's own muscles brings about motion of the part. Active motion is that in which the muscles designed to do the particular movement perform the act.

Passive motion is usually the first step. Active assisted motion, a combination of active and passive, is the second. Active motion is the third; active resisted motion is the fourth, and gives the highest degree of exercise. Exercises are frequently begun with the body horizontal or reclining, then in the sitting position, and finally in the vertical or standing position. Some mechanical aids are invaluable in bridging the gap between inactivity and normal activity. The stationary bicycle for the knees and legs, the rowing machine for the back, the sewing-machine treadle for the feet, marbles for the feet and the collapsible rubber ball for the hands are a few of the simple and more readily available aids. The value of exercise for recovery of function lies not in its vigor or the time given to it, but in the regular and systematic performance of each exercise prescribed. The physician should change the exercise as often as necessary to meet the development of muscles, and someone should supervise it in order for the best results to be obtained.

Local applications of heat before passive or active exercise are valuable in limbering up the part or rendering it less painful. Heat is too often used alone, and employed thus can give only temporary help. The type of heat and the method of application depend upon the physician's choice. While moist heat in the form of hot wet towels applied for a definite period, say twenty minutes, is inexpensive as well as foolproof, dry heat from any of the various infra-red lamps may also have definite usefulness. The heat obtained by diathermy has distinct indications. In applying moist heat, the patient should be warned against the use of electric heating pads, which may become wet and conduct the electric current through the body, with serious results.

Creation of a Favorable Mental Attitude

A condition resembling melancholia is so commonly observed in persons suffering from toxic arthritis that the depth of the depression is often an indication of the patient's general condition, and must be actively treated. It is important to point out to him the favorable developments in his condition, and to stress them in comparison with the unfavorable factors. It is the encouragement which comes from realization of improvement that gives patients the courage to continue the program upon which ultimate recovery depends. Success in developing this mental attitude depends on the physician's ability to apply psychological methods effectively.

Relief of Pain

The treatment of toxic arthritis is too often limited to the relief of pain. Most of the drugs advocated for the disease are intended primarily for that purpose. If this were a self-limiting dis-

ease of short duration, or if the patient tended naturally to recover, the use of pain-relieving drugs would not be objectionable, but under the actual conditions, the use of such medicines is open to serious question. If the measures enumerated under the first four objectives of treatment are effectively carried out, only a minimum of pain-relieving medicine will be required.

Of the many preparations offered for the relief of pain the one of sufficient strength to relieve the pain should be used. But the physician should know the action of the drug on the body as a whole, and should not utilize preparations which, while effective in relieving pain, do serious organic damage to the vital organs. In general, the salicylates and their well-known derivatives should be employed where mild remedies are called for. If it becomes necessary to use excessive quantities, this is evidence that they are not effective, and resort should be had to stronger preparations, even narcotics.

TRAUMATIC ARTHRITIS

Traumatic arthritis may be acute or chronic. The injury resulting from collision with a moving object is likely to be acute, while that resulting from the habitual use of the joint in a faulty manner is likely to be chronic. Acute trauma, unless it causes fracture of the bones entering into the joint, seldom reveals anything by x-ray. Chronic trauma invariably gives rise to hypertrophic changes in and around the joints. Whether the trauma be acute or chronic, rest of the affected joints effectively relieves the symptoms, although one can hardly expect any modification of the hypertrophic changes of chronic irritation.

In the chronic form, the recognition of the causative agent, whether it be faulty posture, excessive abdominal girth or maldevelopment, is essential to the planning of adequate treatment. Control or correction of the traumatizing factors almost always ensures relief of pain and the halting of the arthritic process.

The surgical treatment of chronic arthritis is essentially the restoration of the joint so as to provide some measure of function after the arthritic process itself is no longer active. Surgical procedures during the active stage of the diseases are frowned upon by those who best understand the pathologic physiology of arthritic diseases.

SUMMARY

A new classification of arthritis based on recognizing bacteria, toxins and trauma as essential etiologic factors, is presented.

The treatment of arthritis is outlined. Bacterial arthritis is essentially a surgical disease, and the treatment is governed by surgical principles. The treatment of toxic arthritis represents the main problem of treatment in arthritis, and effective treatment involves: removal of unfavorable factors; restoration of normal physiology; protection of the joints during treatment; creation of a favorable mental attitude; relief of pain. Traumatic arthritis is relieved by removing the traumatic factors.

158 Whitney Avenue

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 24161

PRESENTATION OF CASE

A forty-two-year-old, white, American factory worker entered the hospital with the complaint of hematuria and right-flank pain.

Six days before entry he began to pass grossly bloody urine. The following day he felt somewhat weak and faint and about noon began to have quite severe pain in his right flank which later extended around to the pubis. The pain was accompanied by a burning sensation on micturition and was only temporarily relieved by hypodermic injections. The hematuria and pain continued up to the time of entry, and he frequently passed clots of blood. He had had no previous genitourinary symptoms.

About twenty years before entry he had had a sore on his penis which was cauterized. For a period of two years following that he had had arm and hip injections. He denied ever having had gonorrhea. He had been married for twelve years, and his wife had never been pregnant. His past history and family history were otherwise negative. He had lost no weight and had always considered himself to be in the best of health.

Physical examination revealed a well-developed and nourished man apparently suffering considerable pain. The heart and lungs were negative. The blood pressure was 120 systolic, 80 diastolic. There were tenderness and spasm in the right upper quadrant and right flank, and the entire abdomen was held fairly tight. No masses were made out.

The temperature was 98.6°F., the pulse 100. The respirations were 22.

The urine was grossly bloody, had a specific gravity of 1.020 and contained a trace of albumin but no casts. The sediment of catheter urine specimens showed occasional granular casts and 300 red cells, 5 white cells and many bacteria per high-power field. The blood showed a red-cell count of 3,060,000 with 60 per cent hemoglobin, and a white-cell count of 9400 with 65 per cent polymorphonuclears. The nonprotein nitrogen of the blood was 49 mg. per cent on entry and 29 mg. per cent two days later, the fasting blood sugar was 95 mg. per cent, and the blood chlorides were equivalent to 103 cc. of N/10 sodium chloride. The blood Hinton test was positive, and the blood Wassermann negative. The icteric index was 8, and the van den Bergh was too low to read. A retrograde pyelogram showed abnormal soft-tissue shadows in the right psoas-muscle and right kidney areas. There were no areas of calcification, and the left kidney was not visualized. Injection of the right kidney pelvis and calices revealed intrinsic and extrinsic filling defects. There appeared to be a mass occupying the lower pole of the right kidney which displaced the pelvis upward, so that it was opposite the eleventh rib. The psoas muscle appeared to be obliterated by the mass. A film of the chest showed an unusually high, right diaphragm with a very much limited excursion. There was density in the right lower lobe, and the right costophrenic angle was obliterated. The heart and mediastinum were displaced to the right, and the right lung root was depressed. The aerated portion of the right lung was clear, and the left lung was negative. Three days later an intravenous pyelogram showed somewhat better visualization of the right kidney than previously. The minor calices on the right were slightly dilated, and the pelvis contained multiple irregular filling defects. The outline of the right kidney was clearly seen and appeared to be symmetrically enlarged, with the greatest enlargement in the region of the lower pole and medial aspect of the pelvis. The pelvis and ureter appeared to be displaced laterally. The lower portion of the psoas muscle was visible, but in its upper portion there was an increase in soft-tissue density, which obliterated the outline of the muscle and the kidney. The left kidney pelvis, calices and ureter appeared normal. An x-ray of the chest showed some improvement, with less marked density of the right lower lobe and slightly less elevation of the diaphragm. However, the diaphragm was still very high, and the heart and mediastinum were still displaced to the right of the spine. No fluid was demonstrated in the chest.

On the fourth day the white-cell count was 14,300 with 78 per cent polymorphonuclears, but the temperature remained normal. Two more urine examinations showed from 20 to 50 red cells per high-power field of the sediment.

On the sixth day an operation was performed.

X-RAY INTERPRETATION

DR. AUBREY O. HAMPTON: I saw this patient three days after the onset of his illness, which occurred in another city, and think it should be stressed a little more that he was in a fair degree of shock. He was really ill with the onset of this hemorrhage. We did an intravenous pyelogram at that time, which showed no function of the right kidney and normal function of the left.

All the other findings were about the same as those when he arrived here. I might also add that at that time his abdomen on the right side was rigid.

This retrograde pyelogram shows the displacement of the kidney pelvis upward and laterally as was described, with a large kidney outline — but larger at the lower pole — and with obliteration of the psoas muscle certainly in its upper half. The diaphragm on the right side was, as stated, high and relatively fixed. It is a fairly typical picture of collapse of the right lower lobe, with the fixation of the diaphragm and the general appearance that is usually seen in massive collapse. If he had a tumor of the right lower-lobe bronchus causing collapse of that lobe we should not expect fixation of the diaphragm. Then at examination three days later the lesion in his chest had definitely improved, as had the patient; an intravenous pyelogram done at that time showed function in a previously nonfunctioning kidney, but the filling defects were still present, both inside and outside the kidney pelvis. We thought the inner defects were probably blood clot and the lower defect very likely due to tumor, but we could not explain why the entire right kidney was enlarged and the psoas muscle obliterated, or why the diaphragm was high and fixed.

Differential Diagnosis

Dr. Arthur W. Allen: What are the conditions that could cause hematuria? I suppose we must come down to that. We can fairly safely rule out a systemic disease, because blood was coming from only one kidney. In other words, we are probably dealing with a lesion in or around the right kidney.

A good deal of stress is laid on his probable early syphilis, twenty years before he came in. We have a positive Hinton and a negative Wassermann test. He had had two years of treatment which we assume to be antisyphilitic. Could this be a syphilitic lesion of the right kidney? The only thing that I should consider is the possibility of an aneurysm. It was probably not an aneurysm of the aorta, but I wonder if it could be one of the renal artery. I think the man is pretty young to have an aneurysm, even if he had had syphilis twenty years previously. However, we must keep that possibility in mind.

What neighboring structures could be involved that might cause this difficulty in the region of the right kidney? Could it be some outside influence such as disease of the liver or bowel, or could it be some trouble in the psoas muscle itself? We can rule out actual primary disease of the psoas muscle because the man had had an acute onset only six days prior to entry. So far as I know, deep psoas abscesses or affairs of that sort must be of a chronic nature. This man had lost no weight. Also, I should be inclined to think that the obliteration of the psoas muscle as shown by x-ray was due to the acute episode and was secondary to whatever was the matter with the kidney. If we consider disease of the kidney, we think of the possibility of an aberrant vessel as a cause of hemorrhage and of the possibility of trauma; but we have no history of the latter. If he had had a serious blow in his loin just prior to the onset of his illness we should expect he would show a ruptured kidney with bleeding into the surrounding structures. Could any kind of infection in the kidney produce this picture? It rather seems as though that is not relevant because of the normal white count and temperature. The leukocytosis which developed four days later could be from the absorption of the hematoma which we assume was present about the kidney. Tuberculosis is another cause of kidney damage with blood loss, but this man was too well to have had tuberculosis of the kidney.

We therefore come down to a differential diagnosis of tumor of the kidney, such as hypernephroma or adenocarcinoma that has developed slowly without any effect on this man's general condition and has suddenly burst through its capsule. This sequence of events could explain the acute onset. It could explain the fixed diaphragm, the shadow over the psoas muscle and the hematuria. The only other thing that I can suggest is the possibility of aneurysm based on his old syphilis, and if it were an aneurysm it would have to be aneurysm of the renal vessel. I should expect that if it were aneurysm that there would not be so much obvious tumefaction of the kidney itself and that the kidney would be of normal size: the hematoma about it would, of course, explain the rest of the picture. I believe, however, that this is probably not an aneurysm but a tumor of the kidney, a hypernephroma that has spontaneously ruptured.

Clinical Discussion

Dr. E. Ross Mintz: The question of the type of incision to make was quite a problem. I finally decided to make a retroperitoneal incision, and when I came down to the lumbar dorsal fascia everything seemed to be all right. The moment I incised the fascia and put my hand around the kidney, out came a lot of blood clots. The clots were down in the fossa, and in freeing the kidney at the upper pole, I again ran into a tremendous amount of blood clot beneath the diaphragm. The diaphragm was evidently still pretty high.

The patient had a small renal tumor — an adenocarcinoma — at the lower pole of the kidney. Curiously enough, there were not a great many vessels around the tumor, and I could see no bleeding point that I could definitely say was the origin of the perirenal hematoma. Although the patient was obese and had a lot of perirenal fat, the kidney freed up pretty well. The fat was quite indurated and I cleaned out the entire fossa and removed the kidney. There was no extension of the tumor into the renal vein or neighboring structures.

PREOPERATIVE DIAGNOSIS

Right kidney tumor with retroperitoneal hemorrhage.

DR. ALLEN'S DIAGNOSIS

Tumor of the kidney with rupture of the capsule — probably hypernephroma.

ANATOMICAL DIAGNOSES

Renal-cell adenocarcinoma.
Perirenal hematoma.

PATHOLOGICAL DISCUSSION

DR. TRACY B. MALLORY: The specimen showed a very sharply circumscribed carcinoma at the lower pole of the kidney, as Dr. Mintz described. We found one small eroded vessel in the capsule, not an impressive lesion to explain such massive hemorrhage, but I suppose it must have come from that spot.

DR. HAMPTON: The diffuse enlargement of the kidney is still unexplained because the kidney was not enlarged.

A PHYSICIAN: Both the hematoma and the tremendous amount of fat which extended anteriorly might account for the shadow.

DR. MALLORY: A great deal of the shadow must have been the hematoma.

DR. HAMPTON: That was our conclusion. Heretofore we have believed that if a patient had a ruptured kidney its outline would be obscured by the hemorrhage. We now have to change our point of view, and when we see a large kidney shadow in a case suspected of ruptured kidney, it may be of considerable significance.

A PHYSICIAN: Why were the heart and mediastinum displaced toward the diseased side?

DR. HAMPTON: He had massive collapse of the right lower lobe due to subphrenic irritation.

CASE 24162

PRESENTATION OF CASE

A thirty-four-year-old, white, American laborer entered the hospital with the complaint of right upper quadrant pain of seven months' duration.

Seven months before entry he began to have daily attacks of dull, throbbing pain in the right upper quadrant, just to the right of the umbilicus, which seemed to radiate down to the right iliac crest. The attacks lasted several hours, occurred more often at night, were unrelated to meals, defecation or activity, and were relieved by walking about but not by taking food or soda. The pain was severe enough to interfere with his sleep, but it did not keep him from working. Shortly after the onset his physician took x-rays of his stomach and gall bladder which were reported to be negative. He was put on a bland diet but this had little or no effect on his symptoms. The pain gradually increased in severity until two months before entry, when it became severe enough to force him to stop working. At about the same time he began to have eructations and flatus in increasing amounts. X-rays taken at that time were said to show "duodenitis," and he was put to bed for three weeks on a first-stage and then second-stage Sippy diet. The treatment had almost no effect on the symptoms, and he began to vomit occasionally, which he thought was due to the large amounts of cream. Five weeks before entry he was put back on a normal bland diet. Nausea and vomiting, however, continued once or twice a week and did give him slight relief from his pain. Three weeks before entry his previously regular bowel habits were interrupted, and he became constipated. His stools were lighter in color and his urine became dark. During the week before entry his pain became extremely severe and was occasionally sharp in character but never crampy. He had no definite jaundice, no hematemesis, no bloody or tarry stools and no cardiorespiratory or genitourinary symptoms. He lost 31 lb. in weight in the six weeks before entry. Sixteen years before entry he had had a nervous breakdown for which he was treated for six months with complete relief of symptoms. The past history was otherwise essentially negative, and the family history was not contributory.

Physical examination revealed a well-developed, slightly undernourished man in no acute distress. There was slight pallor of the skin and mucous membranes. The heart and lungs were negative. The blood pressure was 115 systolic, 75 diastolic. The abdomen was somewhat distended and tympanitic. The entire right side of the abdomen and the region over the sigmoid showed voluntary spasm and was tender to even light palpation. The tenderness was most marked just above and to the right of the umbilicus. The liver and spleen could not be felt, and no masses could be made out.

The temperature was 99°F., the pulse 100. The respirations were 20.

The urine examination was negative. The blood showed a red-cell count of 3,500,000 with 55 per

cent hemoglobin, and a white-cell count of 13,600 with 75 per cent polymorphonuclears, 14 per cent lymphocytes, 2 per cent monocytes, 7 per cent eosinophils and 2 per cent basophils. The platelets were normal. The guaiac test on the stool was 3+. The blood Hinton test was negative, and a 1:40,000 tuberculin test negative.

A flat abdominal x-ray film showed several areas of calcification in the right side of the abdomen consistent with calcified mesenteric glands. There were no visible ureteral calculi, the kidneys were normal in size and shape, and there were no unusual soft-tissue masses. Two barium enemas showed no evidence of disease within the colon or terminal ileum. An x-ray of the chest was negative. A gastrointestinal series showed no evidence of lesions in the esophagus or stomach, and the duodenal cap was not deformed. At the junction of the second and third portions of the duodenum there was an upward convex curve with a questionable large area of ulceration in that region. No definite surrounding tumor mass was visible. Hourly follow-up films of the small intestine showed no evidence of disease. A re-examination four days later confirmed the presence of a lesion at the junction of the ascending and descending portions of the duodenum. There was a lobulated tumor mass, with probable ulceration, protruding into the lumen in that area. A Graham test showed very faint filling of the gall bladder, much less than in a normal individual.

The patient continued to have abdominal pain and during the first two weeks in the hospital lost 11 lb. in weight. On the twenty-second day an exploratory laparotomy was performed.

X-Ray Interpretation

Dr. George W. Holmes: The lower films simply show a negative colon and negative chest. In the upper group you can see this very definite deformity of the duodenum. The stomach appears normal. When you study these films more in detail the mucosal pattern is seen to be lost over a considerable area. There is an indentation of the duodenum, which is also somewhat dilated. The appearance in the duodenum is that of a large ulcerated mass. Here are the films of the gall-bladder region. There is a faint shadow of the gall bladder. There is no evidence of stones. It is normal in shape, and the only abnormality is the faintness of the shadow. A shadow like this sometimes occurs when the patient has been fasting before examination; I have seen it happen several times. Similar shadows are also seen in cases with tumor of the pancreas. Most of these cases have very faint or absent gall-bladder shadows. There is no reason to expect that the gall bladder is diseased. The lesion from our point of view should be a tumor of the duodenum, and since I know the answer in this case I will not discuss the possibilities.

Dr. Alfred O. Ludwig: I wonder if it is fair to ask whether by x-ray the lesion was intraduodenal. It was stated in the x-ray report that there was no visible tumor mass outside.

Dr. Holmes: Such a deformity of the duodenum as this I think would be more likely to occur if the lesion were in the duodenum; but one cannot say it did not extend into it from some external organ.

Differential Diagnosis

Dr. Ludwig: Before reading over this case my ignorance on the subject of tumors of the duodenum was almost complete, and the only way I could get anywhere was by studying the literature. So far as the story is concerned it is quite typical of tumor in the duodenum or tumor in the small bowel. Dull pain lasting several hours, of paroxysmal nature, is stressed many times in reports of these cases and is quite characteristic. It is quite interesting that mention is made in a number of these cases of early x-rays that have been said to show duodenitis. Nausea and vomiting, constipation and weight loss are also described as characteristic symptoms of tumors occurring in the duodenum or small bowel. Furthermore, the physical findings — the pallor and anemia, the distended abdomen, and the spasm and tenderness — are characteristic in cases that have tumors in this region. It is interesting that most of the cases described also stress the presence of a palpable mass. That may not have been felt here because it was too small or because of the spasm and rigidity that this patient had. The anemia is of a considerable degree, — 3,500,000 red cells with 55 per cent hemoglobin, — and I think we can say that some of the anemia was certainly due to blood loss from the bowel. The patient had definite occult blood in the stools, although there was no history of any gross bleeding. I was also interested in the eosinophilia of 7 per cent, and it will eventually lead me to make a guess as to the diagnosis.

So far as the differential diagnosis of obstruction in the duodenum is concerned it has been divided according to its source into intraluminal, mural and extraluminal obstruction. The main causes of the intraluminal type are foreign bodies, particularly trichobezoars or hair balls. These are pretty well excluded by x-ray. Probably this case will fall into the mural group, and obstruction may occur from tumors in this region which are benign or malignant. Congenital stenosis of the duodenum also may cause obstruction, but again I believe that such a diagnosis is ruled out by the x-rays. Finally, there is the extraluminal cause of obstruction: from congenital bands; from

adhesions arising from inflammation in neighboring organs, such as the gall bladder; from lesions of the pancreas, usually carcinoma; from enlarged neighboring lymph nodes; and finally there was one case described in which there was obstruction of the third portion of the duodenum from aneurysm of the abdominal aorta directly beneath it.

The diagnosis is narrowed down to a tumor of some sort, which may be either extrinsic or intrinsic. Intrinsic tumors are either benign or malignant, and both are quite rare. The incidence of duodenal carcinoma is 0.03 per cent in a total series of 300,000 or 400,000 autopsies.

Of the malignant tumors, primary carcinoma of the duodenum is most frequent. Next in frequency is carcinoma of the papilla of Vater, next perhaps lymphosarcoma, of which there have been 15 cases reported out of a total series of 84 in the large and small bowels, then spindle-cell sarcomas, and most rarely, leiomyosarcoma of which only 2 were reported up to 1935. Extrinsic malignant tumors must be considered. They arise most commonly in the gall bladder or stomach, but frequently in the pancreas or any other neighboring organ. I do not know whether carcinoids should be classed as malignant tumors; Dr. Mallory could tell us more about that. These are the tumors which on staining show an affinity for silver and are found most commonly in the appendix, but have been reported in the small intestine.

The benign tumors are about half as frequent as the malignant ones. The commonest benign duodenal tumors are the adenomas; these are usually polypoid and arise from mucous cells or from Brunner's glands. The next in frequency are the fibromyomas, and finally a varied group including hemangioma, lymphangioma, neuroma, tumors arising from displaced pancreatic tissue, and so forth.

Most of the reports in the literature stress that all malignant and most benign tumors will bleed, so the presence or absence of bleeding does not help us much. I believe we can rule out carcinoma of the papilla of Vater because of the absence of jaundice. Of course any tumor of the duodenum may eventually cause jaundice by encroaching on the common bile duct, and it may be that the imperfect filling of the gall bladder was due to partial obstruction from the tumor mass. So far as I am concerned, the diagnosis of the nature of this tumor is reduced to a guess, and the only possible clue that I can see is the eosinophilia, which is something we often find associated with Hodgkin's disease or lymphosarcoma. As a pure guess I shall say that the tumor was a lymphoblastoma or lymphosarcoma of the duodenum.

Preoperative Diagnosis

Cancer of duodenum.

Dr. Ludwig's Diagnosis

Primary tumor of the duodenum (lymphoblastoma or lymphosarcoma?).

Anatomical Diagnosis

Carcinoma of duodenum.

Pathological Discussion

Dr. Tracy B. Mallory: This patient was operated on by Dr. Arthur W. Allen, who found a large area of ulceration on the posterior wall of the third portion of the duodenum in approximately the region of the papilla of Vater. He felt from the gross examination that it was probably a tumor and that it had invaded the pancreas. He asked me to make a frozen section from a small piece of the ulcer margin, which he resected after opening the duodenum opposite the crater, but I was unable to find any tumor in it. However, he felt strongly enough that the lesion was malignant not to be unduly influenced by that and proceeded to do a cholecystgastrostomy because he felt certain that sooner or later it would obstruct the common bile duct, and also did a gastroenterostomy to relieve the duodenal obstruction. The later sections of the biopsy showed one minute fragment that was certainly carcinomatous and on the basis of that it was decided a couple of weeks later to make an effort at resection. This required removal of practically the whole duodenum and head of the pancreas, and the operation was made considerably harder by the fact that there was an anomalous insertion of the cystic duct. Normally the distance between the papilla and the point where the cystic duct joins the common bile duct is a matter of several centimeters so that ordinarily it would be possible to do such a resection without cutting the cystic duct. In this case, however, the cystic duct entered the common bile duct only 2 cm. from the papilla, right in the area of tumor involvement; so he was forced to anastomose the cystic duct to the common bile duct in the course of his operation, which prolonged it somewhat. The surgery was completed very successfully; but shock developed, and the patient died the following day. The surgical specimen showed an extensive carcinoma and I cannot say from the specimen whether it was primary in the duodenal wall near the papilla or actually in the papilla. My inclination is to believe that it probably started in the papilla but I think it ulcerated from the start and for that reason never obstructed the bile ducts. At autopsy it was found that there were metastases to one of the regional nodes, so that even if the patient had survived the resection he would not have lived a great many months.

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FOR VALOR

The six members of the Governor's Council, the majority of members of the Massachusetts Medical Society, the members of many other organizations, and the numerous individuals, all interested in the health of the people, deserve well of the Commonwealth of Massachusetts. By their actions or efforts, an attempt to lower the standard of public-health administration in this Commonwealth has been overthrown; and because of this, we herewith offer this citation.

The fact that we have been given this exceptional privilege should make everybody, but especially every doctor, stop and think. Experienced practical politicians will generally admit, when speaking off the record, that when it is organized, the potential vote-getting power of the medical profession is the greatest that exists in any political unit. Its unpredictability, however, is as great as its potential strength. It can be counted on to enforce any measure that is for the ultimate good of its patients, and therefore of the poor and underprivileged. But, because of its uncompromising intellectual honesty, it has the habit of making up its own mind about what the actual rather than the political worth of the proposition in question may be. This is very confusing to the *died*-in-the-wool politician who regards such a matter as the pubic health of the State as of little relative importance compared to party regularity or the "affection of the people" of any community.

Let us not forget this illuminating experience and let us not fail to remind those courageous men who, by their vote, put public welfare above private political expediency that we, as individuals, are grateful: so grateful, that when they again have to speak up for the poor, the sick, the crippled and the dying,—as we have been warned they will,—they can count on our wholehearted backing.

A NATIONAL MEDICAL COUNCIL

News from the recent meeting in Chicago of the Council on Medical Education and Hospitals of the American Medical Association suggests a somewhat new approach toward the solution of the problems confronting the medical profession. It is perhaps primarily a shift in emphasis, and the dominant note struck is "education." References to the address of Dr. Willard C. Rappleye have been made in the lay press, but what has attracted most attention is the suggestion that there be formed a National Medical Council, to be composed of representatives of universities, medical schools, hospitals, the practicing profession, specialist examining boards, state licensing boards and public-health agencies. It is interesting to note that this proposal was made during a discussion of the function of the specialist examining boards at the Congress on Medical Education and Licensure, with representatives of the hospitals participating.

The functions of the proposed council would

be to study the needs of American medicine from the point of view of education, mobilizing the best current opinion regarding the different phases of professional training at its various levels, formulating standards adequate for those levels, and advising universities, hospitals, regulating bodies and governmental agencies on standards, methods and procedures. The end to be attained is better care of the sick: it is the improvement of the preparation of the physician for his peculiar function, from the days of his work as a premedical student until he retires from practice, that is the dominant idea of the new proposal.

There has been developing gradually in this country a widespread opinion that medicine, or speaking more generally the healing art, has been advancing in a too inco-ordinate fashion — a sequel to the remarkable progress in science and the growing general interest in health and ways of promoting it and ways of losing it. It impresses one as a jungle rather than as a well-ordered garden. There is little correlation, great independence of the parts, considerable overlapping and much waste. In fact we ought to have a planned economy, using the word economy in its remote rather than in its immediate sense.

If the plan means the use of intelligence by qualified individuals, the idea meets a favorable reception at once, provided that the authority granted to the body is persuasive rather than coercive. The proposed council would be only advisory in function, and one of the reasons why it has been suggested is fear of increased governmental interference with medical education and medical practice. There can be no doubt that the fear is justified and that there will be continued effort to increase the participation and therefore the control of the government in medicine. This may not be intrinsically wrong, but there are many thoughtful physicians whose opinion is expressed by the well-known saying: "It is worse than a crime; it is stupid." Yet the mere mention of a national medical council arouses antagonisms and fears on the part of many persons, because too often "national" means "national governmental" and we cannot get away from the idea of force or coercion.

There has developed through the past one hundred and fifty years a sense of the unity of these United States. It was one of the elements in the struggle which, according to our point of view, we call the War of the Rebellion or the Civil War or the War between the States. In the World War it was intensified. It grows with the advance in methods of communication and of transportation. In time and space the widely separated North and South and East and West are closer together now than ever before. There has occurred with this development an extension of the intrusion of the national government into many fields. A critical question is, How far shall it intrude? What is a just and wise delimitation of the field for the national government? No one can say offhand, but there is no human intelligence capable of directing our lives wisely from Washington or from any other place on earth.

It is the fundamental democratic dogma that the adult individual is the best guardian of his own rights and the best director of his own life. No matter what qualifications are necessary because of collectivism or socialism or communism, using these words in their general sense and not in their perverted and more frequent acceptation, individualism will endure. It is the infinite worth of the individual that these last 1900 years have been trying to impress upon us. Lord Acton said of liberty: "It is the delicate fruit of a mature civilization." Is there liberty in Socialism or Communism or Nationalism? This is an acid test, devastating if applied to most contemporary organized movements and activities.

A truly national medical council, carried on under the idea that better ways can be found for solving the health problems of the nation and that, in general, people will do what is best for themselves if they know what is best, such a medical council, using science and intelligence and moral suasion rather than coercion, whether direct or indirect, might make a great and significant contribution to our national life. We need to learn

to distinguish between our nation and our government, and to realize how much there is which we do as a nation in which the federal government has properly no part.

A penetrating critic of modern life has said: "The dangers arising . . . are great, particularly in our democratic societies. The directive force of reason is weakened. The leading intellects lack balance. They see this set of circumstances, or that set; but not both sets together. The task of co-ordination is left to those who lack either force or the character to succeed in some definite career." The pertinence of this to governmental direction is perhaps apparent. The problem is, first, to secure, for such co-ordination as is desirable, persons who for sheer ability have succeeded in some definite career; and secondly, to refuse to grant them coercive power, so that they may persuade rather than compel.

OBITUARY

BENJAMIN WHITE

1879-1938

BENJAMIN WHITE was born in Cooperstown, New York, on January 15, 1879. He received a Ph.B. degree from Yale University in 1900 and was appointed Robinson Fellow in the same year. While holding this fellowship he studied in Berlin, Munich, Vienna and London, and in 1903 was given a Ph.D. degree by Yale. During the same year he was made assistant director of the Department of Bacteriology, Hoagland Laboratory, Brooklyn, New York, and in 1907 was appointed director. Because of ill-health he was obliged to go to Saranac, New York, in 1909, and after recovering from his acute illness, worked as an assistant in the Saranac Lake Laboratory until 1911, when he returned to his former position. He also served as consulting bacteriologist to the Long Island College Hospital from 1911 until 1914, when he was appointed assistant director of the Bacteriological Laboratories, New York City Department of Health, and was placed in charge of the biological laboratories at Otisville, New York. In 1920 he was made director of the Division of Biological Laboratories, Massachusetts Department of Public Health, with his chief duty the supervision of the Antitoxin and Vaccine Laboratory at Forest Hills. This position he held until 1933, when he was forced to resign because of ill-health. For the past few years he had lived in New York City, spending most of his time collecting data for and writing a book, *The Biology of Pneumococcus,* which was published this year by the Commonwealth Fund. At the time of his death he was retained by the Commonwealth Fund in a consulting capacity.

When he came to Boston in 1920 he was made a member of the Department of Preventive Medicine and Hygiene, Harvard Medical School, and at the time of his retirement was assistant professor in this department and also in the Department of Bacteriology. In 1920 he was appointed lecturer on biologic assaying and immunology at the Massachusetts College of Pharmacy, and became an associate professor in 1922, which position he held until 1925. Since 1926 he had been a member of the editorial board of the *New England Journal of Medicine,* and during the War had served as a captain in the Sanitary Corps.

He was a fellow of the American Academy of Arts and Sciences, an honorary fellow of the Massachusetts Medical Society and an honorary member of the Aesculapian Club. His memberships included the National Tuberculosis Association, the American Association of Pathologists and Bacteriologists, the Society of American Bacteriologists, the American Public Health Association, the Society of Experimental Biology and Medicine and the American Association of Immunologists.

In 1935 he married Nona Solari, of New York City, and those who knew him intimately realize that the last three years of his life were his happiest. Severely handicapped by a badly damaged heart, he was able to carry on, even spending several months abroad during the past summer; and as a climax, he completed his book and so handed down to posterity a knowledge of intricate facts possessed by few and never before made available to others.

He was an indefatigable worker, and his insistence for detail and accuracy was largely responsible for the many improvements in the manufacture, distribution and use of biological products that had their origin in the Antitoxin and Vaccine Laboratory while he was director. The laboratory itself was a model institution, and the people of Massachusetts received the benefits derived from the distribution and use of biological products of the highest grade.

A great lover of the out-of-doors but necessarily limited in activity on account of health, his favorite avocation was trout fishing. Educated on the Beaverkill and the Esopus, he was an expert with the dry fly, and refused to degrade himself with the lowly worm even when fishing — usually with

little success — in the ice-cold streams of New England during early spring.

His death was a great shock to his friends, who were legion. He certainly made the most out of life, and to many of us there must creep in the hope that we may, within our spans, be able to accomplish as much as he did.

R. N. N.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

M. Fletcher Eades, M.D., *Secretary*
19 Bay State Road
Boston

Case History No. 68. Central Placenta Previa

Mrs. E. K., a thirty-five-year-old multipara in her thirty-sixth week of pregnancy, was referred by her local physician to the prenatal clinic on February 14 because of bleeding.

There was no record of the family history. Her past history was noncontributory except for influenza in 1918. She had had five normal deliveries and one forceps delivery of a stillborn baby, which was said to have been very large. Catamenia began at fourteen, had a twenty-eight-day cycle and lasted five days without pain. Her last period was June 15, making the estimated date of delivery March 21.

Upon physical examination the lungs showed no rales and were resonant throughout. The heart showed no enlargement; there were no murmurs. The blood pressure was 120 systolic, 70 diastolic. The fundus was 27 cm. above the symphysis, and the baby was in LOA position. The urine showed no albumin or sugar.

The present pregnancy had been normal until January 25, when the patient, while trying to vomit, had passed about a pint of blood by vagina. She passed a large clot following this but had no further flow. On February 6 the patient awoke during the night to find that she had had a small amount of bleeding. Since that time she had been in bed and under her physician's care. During the night of February 11 she awoke to find herself in a pool of blood, and bleeding of an undetermined amount continued for about four hours. At that time the patient passed a large clot, and the bleeding stopped. She stayed in bed from that time until her admission to the hospital and had no further complaints except marked thirst. All these spells of bleeding were painless, and there was no evidence of uterine cramps or spasm. There was no abdominal tenderness at any time.

On arrival at the hospital the patient was sent at once to the labor room and a delivery room was set up with a bagging set. Her blood was typed for transfusion. The patient was prepared in the usual manner and examined by the attending physician and the resident. The cervix was patulous and admitted two fingers without difficulty. The placenta could be plainly felt, and on gentle exploration appeared to extend about the whole circumference of the cervix.

As the resident withdrew his fingers, they were followed by a stream of bright blood. A No. 6 bag had already been tested, folded and clamped by uterine forceps. The bag was at once inserted into the cervix and filled with fluid, thereby controlling the hemorrhage.

The patient started shortly in regular labor, and in about two hours the cervix was fully dilated. During labor the blood pressure varied between 100 and 115 systolic, and the pulse rate averaged 92. As soon as the bag was through the cervix, it was removed from the vagina and the baby delivered by internal podalic version. The placenta and membranes came away at once. The baby breathed spontaneously and weighed 6 lb.

On examination the placenta showed a central area covered with dark blood clot.

After delivery the uterus behaved well, and there was no unusual bleeding. The patient was given posterior pituitary extract and ergot, one ampule of each. Ergot by mouth, to maintain firm contraction, was commenced as soon as the patient could retain it. Recovery was uneventful, and mother and baby were discharged well.

Comment. Although painless vaginal bleeding in considerable amount during the last third of pregnancy is always suggestive of placenta previa, positive diagnosis can be made only by inserting the finger through the cervix and feeling the placenta. As in every case where immediate delivery may be necessary, all surgical precautions in the preparation of patient and doctor must be taken.

This case well illustrates what additional preparations are necessary to insure the minimum risk for the mother and baby. If the family physician had tried to diagnose the condition at home, the patient might have died from hemorrhage before reaching the hospital. The patient should have been hospitalized on January 25, when bleeding first occurred. The favorable outcome in this case is no argument for procrastination. It is not safe to allow a patient to bleed two or three times without an attempt's being made to determine the source of the bleeding.

Simply to put a patient to bed who bleeds in the

A series of selected case histories by members of the section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the section.

last months of her pregnancy is unintelligent procrastination. An examination is imperative, and a hospital is the only place where such examination can be intelligently made. It was really good fortune that serious hemorrhage did not occur before this patient reached the hospital. The method of treatment after the patient reached the hospital was ideal.

The baby's risk depends on how extensively the fetal circulation has been damaged by the placental separation, which, in turn, is conditioned, to a large extent, by the site of placental implantation; the method of delivery is of secondary importance.

LEGISLATIVE NOTES

On April 1 the Committee on State and National Legislation issued a new Legislative Bulletin which will be sent to any fellow on request.

Since April 1 the following changes in status have taken place in the bills in which we are interested:

House Bill 39, relative to the definition of the terms "rendering medical service," "practice of medicine" and "holding one's self out as a practitioner of medicine" and its companion bill House Bill 40, exempting certain classes, such as dentists, optometrists and so forth, from the above bill, have been voted "no legislation necessary" by the Legislature.

House Bill 1529, which provided for the furnishing by the Commonwealth of free oxygen tents to the hospitals in cities of over 30,000 population, has been given "leave to withdraw" by the Legislature, as has House Bill 1650 which was an act changing the charter of Middlesex University.

House Bill 758, which was a bill to postpone the operation of the statute raising the standards of medical education from January 1, 1939, to January 1, 1942, has been redrafted and is now to be known as House Bill 1845. The new bill postpones the action for two years instead of three years and has been passed in the House.

House Bill 456, which provided for a state fund to replace the present workman's compensation law, has been given "leave to withdraw" by the Legislature.

House Bill 1084, which provided that the restrictions in regard to graduates of a poorer grade of medical schools should be lowered, has been given "leave to withdraw," and this action has been approved by the Senate.

Of House Bill 1279 and Senate Bill 361, both of which provided for the issuance of certificates of approval for bacteriological laboratories, the former has been "referred to the next annual session" and the latter given "leave to withdraw" by the House. Final action by the Senate, however, has not been taken.

The bill for the annual registration of physicians, House Bill 41, has been voted "no legislation necessary" by the House. It will now go to the Senate.

House Bill 42, relative to the suspension or revocation of certificates to practice medicine, has been passed to be engrossed. We favored this bill.

Of the fifty-six bills in which the Committee on State and National Legislation is interested, which were before the current Legislature, thirty-one have been disposed of for this session.

APPLICANTS FOR FELLOWSHIP

PUBLISHED IN ACCORDANCE WITH RESOLUTIONS ADOPTED AT THE ANNUAL MEETING OF THE COUNCIL JUNE 2, 1937

BARNSTABLE DISTRICT

RICE, JACKSON M., 464 Main Street, Hyannis.
Middlesex College of Medicine and Surgery, 1931.

Submitted by John I. B. Vail, *Secretary*.

BERKSHIRE DISTRICT

BLAIS, WILFRED A., 186 Elm Street, Pittsfield.
McGill University Faculty of Medicine, 1935.

MACEK, JAMES J., 4 Centre Street, Adams.
Tufts College Medical School, 1936.

ROBERTS, PERCIE, Sheffield.
Middlesex College of Medicine and Surgery, 1929.

SHIBEL, EDWARD E., 205 East Main Street, North Adams.
Georgetown University School of Medicine, 1934.

Submitted by Hugh J. Downey, *Secretary*.

BRISTOL SOUTH DISTRICT

BARRETT, JAMES A., 227 Union Street, New Bedford.
Maryland Medical College, 1910.

LECLAIR, HORMIDAS H., 227 Union Street, New Bedford.
Tufts College Medical School, 1914.

Submitted by Charles Shanks, *Secretary*.

ESSEX NORTH DISTRICT

EWELL, JOHN W., The Close, Rowley.
Harvard Medical School, 1936.

ODDY, PARKINSON L., 477 Essex Street, Lawrence.
Middlesex College of Medicine and Surgery, 1932.

QUIGLEY, THOMAS J., 102 Main Street, Andover.
Boston University School of Medicine, 1928.

ST. LOUIS, LIONEL E., 359 Haverhill Street, Lawrence.
Middlesex College of Medicine and Surgery, 1931.

SZOSTAK, RAYMOND G., 250 Prospect Street, Lawrence.
Kansas City University of Physicians and Surgeons, 1931.

Submitted by Elmer S. Bagnall, *Secretary*.

ESSEX SOUTH DISTRICT

ALEXANDER, HAROLD G., Beverly Hospital, Beverly.
Tufts College Medical School, 1937.

GREEN, HARRY, 31 Middle Street, Gloucester.
Massachusetts College of Osteopathy, 1929.
Middlesex College of Medicine and Surgery, 1932.

HOPKINS, STANFORD W., 7 South Street, Marblehead.
Harvard Medical School, 1933.

MERRILL, EVERETT A., 3 Lewis Street, Lynn.
Tufts College Medical School, 1913.

PATTERSON, GEORGE W., 44 Lewis Street, Lynn.
College of Medical Evangelists, 1935.

Submitted by Ralph E. Stone, *Secretary*.

FRANKLIN DISTRICT

FOOTNICK, SAMUEL, 11 Prospect Street, Orange.
Middlesex College of Medicine and Surgery, 1930.

Submitted by Charles Moline, *Secretary*.

HAMPDEN DISTRICT

BACHMANN, RICHARD, 121 Chestnut Street, Springfield.
University of Munich, 1921.

BOURGEAULT, OSCAR A., 127 Chestnut Street, Holyoke.
Georgetown University School of Medicine, 1936.

COEN, WILLIAM B., 1214 Main Street, Springfield.
Tufts College Medical School, 1935.

DIXON, STANLEY R., 698 Alden Street, Springfield.
Missouri College of Medicine and Science, 1927.

FEDERICI, LOUIS M. N., 76 Woodside Terrace, Springfield.
College of Physicians and Surgeons, Boston, 1923.

KOTARSKI, WILLIAM C., 700 Chestnut Street, Springfield.
Middlesex College of Medicine and Surgery, 1929.

MERICAN, MILTON, 225 Chestnut Street, Holyoke.
St. Louis College of Physicians and Surgeons, 1924.

NIEMIRO, BERNARD J., 80 Elm Street, Westfield.
Middlesex College of Medicine and Surgery, 1931.

SHERMAN, DAVID E., 447 Sumner Avenue, Springfield.
Middlesex College of Medicine and Surgery, 1930.

TETA, FRANCIS A., 1214 Main Street, Springfield.
Middlesex College of Medicine and Surgery, 1927.

Submitted by Hervey L. Smith, *Secretary*.

HAMPSHIRE DISTRICT

HUFFMIRE, JOHN A., Main Street, Huntington.
Middlesex College of Medicine and Surgery, 1924.

LAWN, HAROLD J., United States Veterans' Association Facility No. 95, Northampton.
University of Minnesota Medical School, 1935.

MAKOFSKI, ANTHONY B., 15 King Street, Northampton.
Kansas City University of Physicians and Surgeons, 1929.

MCKEOUGH, WILFRED A., Hampshire County Sanatorium, Haydenville.
Tufts College Medical School, 1924.

WARNER, LULU H., Belchertown State School, Belchertown.
Tufts College Medical School, 1936.

Submitted by Joseph D. Collins, *Secretary*.

MIDDLESEX EAST DISTRICT

KIRKWOOD, SAMUEL B., 5 Swan Road, Winchester.
Harvard Medical School, 1931.

MACDOUGALL, ERNEST C., 117 Church Street, Wilmington.
Massachusetts College of Osteopathy, 1929.
Middlesex College of Medicine and Surgery, 1931.

MAIETTA, ANGELO L., 408 Main Street, Winchester.
Middlesex College of Medicine and Surgery, 1930.

MURRAY, JOHN M., 9 Sheffield West, Winchester.
University of Pennsylvania School of Medicine, 1921.

Submitted by Kenneth L. Maclachlan, *Secretary*.

MIDDLESEX NORTH DISTRICT

LANDAU, IRVING I., Andover Road, Billerica.
University of Berlin, 1934.

STEWART, ARTEMAS J., 124 Luce Street, Lowell.
Harvard Medical School, 1933.

Submitted by Theodore A. Stamas, *Secretary*.

MIDDLESEX SOUTH DISTRICT

BONNAR, JAMES M., JR., 775 Trapelo Road, Waltham.
Columbia University College of Physicians and Surgeons, 1935.

BROWN, FELIX M., 163 Hillside Avenue, Arlington.
Vanderbilt University School of Medicine, 1924.

BUTTERS, CHARLES M., 81 Hudson Street, Somerville.
Tufts College Medical School, 1937.

CHOPE, HAROLD D., 123 Mt. Vernon Street, Newtonville.
Stanford University School of Medicine, 1931.

CINCOTTI, JOHN J., 442 Pleasant Street, Belmont.
Boston University School of Medicine, 1935.

CROOKER, JAMES L., 386 Broadway, Somerville.
Middlesex College of Medicine and Surgery, 1930.

EWERT, EARL ERNST, 7 Longfellow Road, Cambridge.
University of Illinois College of Medicine, 1927.

GOLDBERG, BERNARD, 1242 Commonwealth Avenue, Allston.
University of Berlin, 1935.

HADDEN, FREDERICK C., 27 Walnut Street, Natick.
University of Minnesota Medical School, 1934.

HARTWELL, CONSTANCE G., Fernald State School, Waverley.
Boston University School of Medicine, 1935.

HOOD, WILFRED T., 7 Felton Street, Hudson.
Tufts College Medical School, 1935.

HOOPER, FRANCIS H. W., 59 Adams Street, Somerville.
Boston University School of Medicine, 1936.

HOUGHTON, JOHN D., 152 Suffolk Road, Chestnut Hill.
Harvard Medical School, 1932.

HOWARD, ERNEST B., 97 Chester Street, Allston.
Boston University School of Medicine, 1936.

HUNT, REGINALD S., 20 Birch Hill Road, Newtonville.
Boston University School of Medicine, 1935.

ISENMAN, ROBERT, 356 Ferry Street, Malden.
Tufts College Medical School, 1930.

JACOBSON, BERNARD M., Massachusetts General Hospital, Boston. (Residence: Newton Centre.)
Harvard Medical School, 1929.

KALLAN, ALPHONSE C., 929 Commonwealth Avenue, Newton Centre.
McGill University Faculty of Medicine, 1935.

KOWAL, SAMUEL J., 215 Harvard Avenue, Allston.
Boston University School of Medicine, 1936.

LEVINE, HARRY, 891 Massachusetts Avenue, Cambridge.
Middlesex College of Medicine and Surgery, 1925.

MCLEAN, EMORY A., 16 Curtis Street, West Somerville.
Cornell University Medical College, 1934.

MULLEN, GEORGE T., 1783 Massachusetts Avenue, Cambridge.
Boston University School of Medicine, 1932.

PAVONE, ROCCO, 192 Crafts Street, Newtonville.
Tufts College Medical School, 1936.

PIJOAN, MICHEL, 24 Chestnut Hill Terrace, Chestnut Hill.
Johns Hopkins University School of Medicine, 1934.

PLUNKETT, RICHARD J., 128 Mt. Auburn Street, Watertown.
Tufts College Medical School, 1933.

RABINOWITZ, JAMES I., 1284 Commonwealth Avenue, Allston.
Tufts College Medical School, 1932.

RAVREBY, LOUIS, 330 Mt. Auburn Street, Cambridge.
Boston University School of Medicine, 1936.

RICHTER, HARRY J., 1056 Commonwealth Avenue, Boston.
University of Michigan Medical School, 1932.

RICKLESS, HERMAN, 111 Fellsway West, Medford.
Tufts College Medical School, 1930.

SAUNDERS, GORDON A., 47 Horne Road, Belmont.
Harvard Medical School, 1935.

STILLMAN, JAMES S., 164 Elgin Street, Newton Centre.
Harvard Medical School, 1934.

TAURO, ARTHUR L., 53 Fellsway West, Medford.
Boston University School of Medicine, 1936.

WALSH, JAMES R., 189 Concord Street, Framingham.
University of Pittsburgh School of Medicine, 1928.

WHITE, SEYMOUR J., 17 Washington Street, Malden.
University of Paris Medical School, 1936.

Submitted by Alexander A. Levi, *Secretary*.

NORFOLK DISTRICT

AISNER, MARK, 36 Fessenden Street, Mattapan.
Tufts College Medical School, 1935.

BALABAN, JOSEPH S., 392 Broadway, South Boston. (Residence: Brookline.)
University of Constantinople, 1919.

BETTS, REEVE H., 1101 Beacon Street, Brookline.
Harvard Medical School, 1933.

BIGELOW, ROBERT B., 302 Adams Street, Milton.
University of Michigan Medical School, 1932.

BIGURIA, FERNANDO, 214 Riverway, Roxbury.
National University, Guatemala City, C. A., 1929.

CARNICELLI, THOMAS J., Exchange Street, Millis.
Tufts College Medical School, 1934.

CHRISTIAN, HENRY A., 1731 Beacon Street, Brookline.
Johns Hopkins University School of Medicine, 1900.

CIVEN, EVA, 1077A Blue Hill Avenue, Dorchester.
Middlesex College of Medicine and Surgery, 1926.

CONE, GERTRUDE, 200 Minot Street, Dorchester.
Tufts College Medical School, 1937.

CONNOR, JOHN H. F., State School, Wrentham.
College of Physicians and Surgeons, Boston, 1907.

DIRAGO, JOSEPH V., 44 York Terrace, Brookline.
Middlesex College of Medicine and Surgery, 1932.

DOLAN, RALPH F., 529 Gallivan Boulevard, Dorchester.
Tufts College Medical School, 1931.

DONLEY, DOROTHY E., Boston State Hospital, 591 Morton Street, Dorchester Center.
Vanderbilt University School of Medicine, 1930.

EDDY, AUGUSTINE W., 82 Walpole Street, Norwood.
Tufts College Medical School, 1932.

EGAN, WILLIAM J., 20 Richfield Street, Dorchester.
Harvard Medical School, 1936.

FINKELSTEIN, SAMUEL M., 72 Edson Street, Dorchester.
Middlesex College of Medicine and Surgery, 1931.

FLAKE, CARLYLE G., 300 Longwood Avenue, Roxbury.
University of Virginia Department of Medicine, 1931.

FRIEDGOOD, HARRY B., 25 Shattuck Street, Roxbury.
Johns Hopkins University School of Medicine, 1928.

GETTING, VLADO A., 11 Perkins Square, Jamaica Plain.
Harvard Medical School, 1935.

GLASSMAN, NATHAN B., 1039 Blue Hill Avenue, Dorchester.
College of Physicians and Surgeons, Boston, 1916.

GLODT, MILTON, 19 Thatcher Street, Brookline.
University of Basel, Switzerland, 1936.

HELWIG, ELSON B., 179 Kent Street, Brookline.
Indiana University School of Medicine, 1932.

HENDRIX, OLIN C., 249 River Street, Mattapan.
Harvard Medical School, 1929.

JOHNSON, PAUL REVERE, 11 Evans Road, Brookline.
Tufts College Medical School, 1932.

KIMMEL, CHARLES B., 368 Longwood Avenue, Roxbury.
Northwestern University Medical School, 1933.

LIA, BIANCA R., New England Hospital for Women and Children, Roxbury.
University of Pittsburg School of Medicine, 1932.

LYONS, CHAMP, 26 Pilgrim Road, Wellesley.
Harvard Medical School, 1931.

MAHER, JOSEPH P., 238 Walnut Street, Dedham.
Tufts College Medical School, 1935.

MURPHY, LAURENCE J., 53 Stockton Street, Dorchester.
Tufts College Medical School, 1936.

NICKERSON, DONALD A., Centre Street, Dover.
Tufts College Medical School, 1933.

PARSONS, EVELYN L., 326 Washington Street, Wellesley Hills.
Boston University School of Medicine, 1932.

REESE, CHARLES A., 249 River Street, Mattapan.
Harvard Medical School, 1906.

RINKEL, MAX, 439 Washington Street, Brookline.
Christian Albrechts University Medical School, Kiel, 1926.

ROSEN, HENRY, 128 Sewall Avenue, Brookline.
Tufts College Medical School, 1936.

RUBY, SYLVIA, 60 Beals Street, Brookline.
Woman's Medical College of Philadelphia, 1935.

SHENKER, HAROLD L., Main Street, West Medway.
Kansas City University of Physicians and Surgeons, 1932.

SOLOMON, PHILIP, 58 Wolcott Road, Brookline.
Harvard Medical School, 1930.

STEWART, HAROLD L., 195 Winthrop Road, Brookline.
Jefferson Medical College of Philadelphia, 1926.

SULLIVAN, CHARLES L., Boston Lying-in Hospital, Roxbury.
Boston University School of Medicine, 1935.

WENZLER, FRANCIS J., 385 Columbia Road, Dorchester.
Tufts College Medical School, 1934.

WEST, FRANCIS J., 22 Alteresko Avenue, Dorchester.
Harvard Medical School, 1935.

Submitted by Frank S. Cruickshank, *Secretary*.

NORFOLK SOUTH DISTRICT

DAVIS, GEORGE W., Quincy City Hospital, Quincy.
University of Rochester School of Medicine, 1934.

DURANTE, FRANK, 686 Broad Street, East Weymouth.
Middlesex College of Medicine and Surgery, 1931.

MCGOWAN, JOHN M., 1245 Hancock Street, Quincy.
Dalhousie University Faculty of Medicine, 1933.

SHANNON, PAUL J., 106 Central Street, Hingham.
Middlesex College of Medicine and Surgery, 1930.

SHEFFNER, SIDNEY A., 8 Dartmouth Street, Quincy.
Tufts College Medical School, 1930.

SOLOMON, SAMUEL, 452 Washington Street, Quincy.
Middlesex College of Medicine and Surgery, 1932.

Submitted by Robert L. Cook, *Secretary*.

PLYMOUTH DISTRICT

FORTUNOW, JACOB J., Main Street, Marshfield.
Middlesex College of Medicine and Surgery, 1930.

GOLDEN, JOSEPH F., 85 Park Street, Stoughton.
Tufts College Medical School, 1911.

KELLY, ALBERT L., 37 Brockton Avenue, Abington.
Middlesex College of Medicine and Surgery, 1932.

MILLER, ROBERT T. JR., Duxbury.
Johns Hopkins University School of Medicine, 1903.

PARISH, FRED A., 191 South Avenue, Whitman.
Middlesex College of Medicine and Surgery, 1931.

Submitted by Fred F. Weiner, *Secretary*.

SUFFOLK DISTRICT

BRADLEY, JOSEPH J., Boston City Hospital, Boston.
Boston University School of Medicine, 1935.

DECKER, BRIANT L., 462 Park Drive, Boston.
Harvard Medical School, 1930.

DEUTSCH, EMMANUEL, Boston City Hospital, Boston.
Tufts College Medical School, 1935.

FRANKMAN, WILLIAM, 362 Commonwealth Avenue, Boston.
St. Louis College of Physicians and Surgeons, 1921.

GOODBERG, BURTON C., 818 Harrison Avenue, Boston.
Boston University School of Medicine, 1935.

HEELS, GEORGE E., Long Island Hospital, Boston Harbor.
Harvard Medical School, 1932.

HOMMEL, EDWARD H., 186 Dorchester Street, South Boston.
Middlesex College of Medicine and Surgery, 1931.

KAMENS, ISRAEL M., 109 Washington Avenue, Chelsea.
Boston University School of Medicine, 1935.

LARSEN, CARL C., 657 East 5th Street, South Boston.
Tufts College Medical School, 1933.

MALOOF, FREDERIC G. F., 204 Huntington Avenue, Boston.
Middlesex College of Medicine and Surgery, 1931.

MARTIN, SAMUEL F., 243 Charles Street, Boston.
Harvard Medical School, 1934.

PIERCE, FRANK R., Long Island Hospital, Boston.
Harvard Medical School, 1934.

POLLOCK, HENRY M., JR., Boston City Hospital, Boston.
Boston University School of Medicine, 1936.

ROIFF, HARRY S., 159 Shurtleff Street, Chelsea.
St. Louis College of Physicians and Surgeons, 1923.

SULKOWITCH, HIRSH W., 109 Mt. Vernon Street, Boston.
Johns Hopkins University School of Medicine, 1932.

VILKER, ARTHUR H., 1510 North Shore Road, Revere.
Middlesex College of Medicine and Surgery, 1930.

Submitted by John P. Monks, *Secretary*.

WORCESTER DISTRICT

CAMERON, DONALD E., Worcester State Hospital, Worcester.
Glasgow University, 1924.

EDWARDS, HUGH S., Worcester County Sanatorium, Worcester.
Medical College of Virginia, 1935.

KANEB, GEORGE D., 3 Germain Street, Worcester.
McGill University Faculty of Medicine, 1936.

LORGE, HEINZ, Rutland State Sanatorium, Rutland.
University of Frankfurt, 1933.

MAHONEY, JOSEPH A., 700 Pleasant Street, Worcester.
Kansas City University of Physicians and Surgeons, 1926.

MCCABE, GEORGE E., The Memorial Hospital, Worcester.
Tufts College Medical School, 1936.

MEYERS, JACK, 771 Main Street, Worcester.
Boston University School of Medicine, 1936.

O'BOYLE, THOMAS J., 228 Main Street, North Brookfield.
Middlesex College of Medicine and Surgery, 1928.

RUSSELL, FRANK H., 78 Burncoat Street, Worcester.
University of Tennessee College of Medicine, 1917.

SULLIVAN, EDWARD C., 6 Stoneland Street, Worcester.
Middlesex College of Medicine and Surgery, 1929.

TANNENBAUM, ISRAEL S., 985 Main Street, Worcester.
Kansas City University of Physicians and Surgeons, 1929.

VESTAL, TOM F., Worcester County Sanatorium, Worcester.
University of Maryland School of Medicine and the College of Physicians and Surgeons, 1929.

WOLBARSHT, ABRAHAM, Worcester Hahnemann Hospital, Worcester.
Tufts College Medical School, 1937.

Submitted by Erwin C. Miller, *Secretary*.

WORCESTER NORTH DISTRICT

WHEELER, CHARLES A., 106 West Street, Leominster.
Harvard Medical School, 1935.
Submitted by Francis M. McMurray, *Secretary*.

MEDICAL POSTGRADUATE EXTENSION COURSES

The following sessions, given by the Massachusetts Medical Society in co-operation with the Massachusetts Department of Public Health, the United States Public Health Service and the Federal Children's Bureau, have been arranged for the week beginning April 25:

BARNSTABLE

Sunday, May 1, at 4:00 p. m., at the Cape Cod Hospital, Hyannis. Subject: The Use and Misuse of Prontylin. Instructor: R. Cannon Eley. John I. B. Vail, *Chairman*.

BERKSHIRE

Thursday, April 28, at 4:30 p. m., at the House of Mercy Hospital, Pittsfield. Subject: The Use and Misuse of Prontylin. Instructor: Benjamin W. Carey, Jr. Melvin H. Walker, Jr., *Chairman*.

BRISTOL SOUTH (Fall River Section)

Monday, April 25, at 4:30 p. m., at the Union Hospital, Fall River. Subject: Toxemias of Pregnancy. Instructor: Joseph W. O'Connor. Howard P. Sawyer and Robert H. Goodwin, *Chairmen*.

ESSEX NORTH

Friday, April 29, at 4:30 p. m., at the Lawrence General Hospital, Lawrence. Subject: Toxemias of Pregnancy. Instructor: James C. Janney. John Parr, *Chairman*.

FRANKLIN

Wednesday, April 27, at 8:00 p. m., at the Franklin County Hospital, Greenfield. Subject: Early Syphilis. Instructor: Francis M. Thurmon. Halbert G. Stetson, *Chairman*.

HAMPDEN

Thursday, April 28, at 4:00 p. m., at the Academy of Medicine, Professional Building, 20 Maple Street, Springfield, and at 8:00 p. m., in the Outpatient Department of the Skinner Clinic, Holyoke Hospital, Holyoke. Subject: The Use and Misuse of Prontylin. Instructor: R. Cannon Eley. George D. Henderson and George L. Schadt, *Chairmen*.

HAMPSHIRE

Wednesday, April 27, at 4:15 p. m., in the Nurses' Home, Cooley Dickinson Hospital, Northampton. Subject: Some Complications of Labor: Analgesics in Labor: Instructor: James C. Janney. Warren P. Cordes, *Chairman*.

MIDDLESEX SOUTH

Wednesday, April 27, at 4:00 p. m., at the Cambridge Municipal Hospital, Cambridge Street, Cambridge. Subject: Drug Therapy in Pediatrics. Instructor: Warren R. Sisson. Edmund H. Robbins, *Chairman*.

THE MASSACHUSETTS CANCER PROGRAM

Dr. Chadwick: A year has passed since I have had an opportunity to discuss the Massachusetts Cancer Program with you. At that time a report of the accomplishments of the first ten years was presented. Tonight the achievements of another year will be demonstrated. And yet, these remarkable incidents can scarcely be called the results of any single year. A program that is so thoroughly integrated among all the individuals in the State gains momentum as it progresses, and each year represents the cumulation of all preceding years rather than the limited results of the single year in question. This month is "Cancer Month" throughout the United States. Massachusetts is co-operating by making public the regular activities of the State Cancer Program, for this State has twelve cancer months. For example, during this month of April there will be, in 21 strategic points throughout the State, 76 state-aided cancer clinics at which there will be an average attendance of 20 physicians and an average attendance of 18 patients. At each of these 21 cities, in place of one of the regular clinics, will be a cured-cancer clinic at which cured cancer patients have volunteered to present themselves to show what has been done for them and what may be done for others. On April 1, for example, 18 cured cases were presented at the Springfield clinic, 10 were presented at the Greenfield clinic, and yesterday 30 were presented at Fitchburg and 5 at Newburyport. More than 300 cured cases will be presented altogether throughout the State. The other clinic communities in which cured cases will be shown are Boston, — at the Beth Israel Hospital and the Boston Dispensary, — Brockton, Fall River, Gardner, Gloucester, Hyannis, Lawrence, Lowell, Lynn, New Bedford, North Adams, Northampton, Pittsfield, Worcester, Pondville and Westfield. There is hardly a residence in the State more than twenty-five miles distance from one of these centers. This new presentation of these 21 cured-cancer clinics in these points near every home will bring the fact of the curability of cancer close to every person. Many of us recognize that most cancer is curable if treated early, but to see individuals who have had the disease and now are free from it and have had no sign of a recurrence for at least five years, is a comforting and reassuring sight.

Dr. Lombard: Before you mention the other parts of the regular program that are being publicized this month, will you explain what constitutes "cured cancer" in the eyes of physicians conducting these clinics, and the number of cases that may be justifiably considered to be cured cancer cases?

Dr. Chadwick: A physician speaks of a cured cancer case as any individual who once had the disease and has been free from it for at least five years. This period of time is chosen because if there is going to be a recurrence of the disease, it usually occurs within this interval. Therefore, the only cures obtained at our cancer clinics that can be considered are those that came between 1927 and 1933. More than 1000 individuals are included in this group. This group of cured cases from our clinics does not begin to measure all the cured cancers that have occurred within this same period, as only a small percentage of the individuals with cancer go to the Massachusetts Cancer Clinics. It is a conservative estimate that approximately 1000 individuals are being cured annually in this State. Will you tell of the call that came to your office recently concerning a cured cancer?

Dr. Lombard: That call was a very interesting one, and is, in a way, symbolic of what is possible in this State. The woman who phoned said she was aware of the work that the State was doing and wanted to help. She said her mother had been cured of a cancer fourteen years before and was now ninety years old, and was well and active. All the records of the case had been collected by these two public-spirited women, including the original pathological slide with its cancer tissue. The woman who had been cured volunteered to appear at any of the cured-cancer clinics or to help in any way she could to publicize the curability of cancer. This demonstrates again the basic trait of the people of our Commonwealth — a sense of community responsibility, a willingness to work, without reward or recognition, for the common good. What other activities are going on in connection with the Cancer Program this month?

Dr. Chadwick: The tumor diagnostic service will receive specimens suspected of being cancerous from approximately 275 physicians. These specimens will be examined immediately upon their receipt and a detailed report will be despatched without delay to the physician who sent the specimen. This is a service that is conducted at no cost to physician or patient. The Pondville Hospital has 140 beds for the care of patients with cancer. The Westfield State Sanatorium has a 50-bed capacity for the care of cancer patients. The statistical or research section of the cancer division will continue its several studies on the various aspects of the disease. The *New England Journal of Medicine* will publish an important contribution to the literature from this department on the "Hereditary Aspects of Cancer," based on years of research. Two hundred and seventy-five of the 355 communities in Massachusetts, which have already been enrolled in the Co-operative Cancer Control Committee Program, will hold more than 1000 group meetings attended by nearly 50,000 persons, and addressed by more than 1000 local physicians. This, in brief, is the program of "Cancer Month" in Massachusetts — a State of nearly 4,500,000 inhabitants, nearly 7000 physicians, and 355 communities. Are there any measures to show just how effective this State Cancer Program really is?

Dr. Lombard: The most effective measure of the significance of the Massachusetts Cancer Program is the concrete evidence that more individuals are going to their physicians as soon as they notice an abnormality. The percentage of individuals who sought the advice of their physicians within the first month of the disease has increased from 12 per cent in 1933 to 18 per cent in 1937. The average delay of all patients has decreased from more than six months in the first nine years of the program to five months in 1936 and 1937. To be sure, five months is a long period for an individual with cancer to delay

A "Green Lights to Health" broadcast given by Dr. Henry D. Chadwick and Dr. Herbert L. Lombard on Wednesday, April 6, and sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

before consulting a physician, but it is far better than the figure of more than six months. The physicians of Massachusetts are taking the lead in referring patients to the cancer clinics. In the early days of the movement, many patients came to the clinics because of newspaper publicity. The percentage referred by physicians was relatively small. Today, this has entirely changed. Nearly 90 per cent of all cancer patients who are attending the Massachusetts cancer clinics are referred by their physicians. The teaching clinics have had a great deal to do with this change in the referring of patients by physicians. Will you explain the development of these clinics?

Dr. Chadwick: Teaching clinics were instituted in 1933. They are conducted in the same locality and at the same time as one of the regular clinics. The local medical profession invites to these special clinics some individual physician, whose specialty is cancer; he meets with the local group and discusses from his experience improved means of diagnostic procedure and treatment. The number of such clinics has increased from 2 in 1933 to 69 in 1937. In the first year, only 75 physicians attended these clinics. In 1937, 1384 physicians were in attendance. This enormous increase is another index of the success of the Cancer Program. Has there been an increase in the number of individuals with cancer coming to the clinics?

Dr. Lombard: The total individuals with cancer attending the clinics has steadily increased. In the first year there were 302; in 1937 there were 1319. The total attendance at the cancer clinics in 1937, including new and old cancer patients, as well as non-cancer patients, was 12,454. Of the new patients, about one third had cancer. Of the old patients, a much larger percentage had cancer. Of the group of 302 cancer patients who came in 1927, 24 per cent are alive today.

Each part of the cancer program that has been discussed so far has depended for its success on the mutual efforts of the medical profession, the public-health profession, and the laity. Of all the underlying parts of the program, which do you consider the most completely dependent on the individual co-operation of every person in the Commonwealth and the most far-reaching in end result?

Dr. Chadwick: Without any hesitation, I feel that our state-wide educational plan, which we call advisedly the Co-operative Cancer Control Committee Program, is the cornerstone of the Massachusetts Cancer Program. This program for the dissemination of information concerning cancer consists in the teaching of exact knowledge by an authority in the person of the family physician to the individual as one of a small group. To accomplish this in a state of 355 communities with a population of nearly 4,500,000 and nearly 7000 physicians, each community in the State has, or is in the process of having, a Co-operative Cancer Control Committee. The individual committees are integral parts of the state-wide Co-operative Cancer Control Program. Two hundred and seventy-five of the communities in the state are organized at the present time. These committees differ from many other educational committees in that they are not selective and consequently restricted to limited groups or classes, but are rather inclusive in nature and represent every type of group and individual in the community. Over 6000 representatives have agreed to serve on these committees and to urge their respective clubs or groups to have annual talks on cancer given by local physicians. The records show that over 100,000 individuals in this State were reached with cancer talks during 1937.

Dr. Lombard: This educational program, which began in Massachusetts and is being watched with interest all over the world, seems to be direct and simple in plan. Why do you think it has been so much more successful than other educational plans for the control of this disease?

Dr. Chadwick: The essence of the success of the educational methods of the Massachusetts Cancer Program lies in its inherent sharing of known facts about cancer by a generous and informed profession with a co-operative and receptive public. Fears are not stressed; they are rationalized. Symptoms are not presented as the basis of exact knowledge; they are merely alluded to in the sane discussion of the larger problem. The physician, with his increasing interest in the progressive steps in the knowledge of cancer, the disease, presents the subject simply and intelligibly. The public, responding as an individual would respond to a real discussion of an absorbingly interesting subject, rises to the mental challenge of this approach. As a result, all classes, groups, races and professions are united in this basic co-operative and successful program which disseminates exact knowledge concerning cancer. This engenders a realization of the need for prompt action. Exact knowledge and prompt action effect control.

RECENT DEATHS

BULIAN — Moses Bulian, M.D., of 274 Seaver Street, Roxbury, died November 17, 1937. He was in his fifty-third year.

A native of Russia, he received his degree from Kiev Medical Institute in 1912. He was a fellow of the American Medical Association and the Massachusetts Medical Society.

His widow survives him.

COTTON — Frederic Jay Cotton, M.D., of 239 Beacon Street, Boston, died April 14. He was in his sixty-ninth year.

Born in Prescott, Wisconsin, he was graduated from Harvard University in 1890 and received his degree from Harvard Medical School in 1894. Following his graduation he was for several years house surgeon at the Massachusetts General Hospital, after which he studied at the University of Vienna. He also attended the College of Physicians and Surgeons in New York City. For several years Dr. Cotton was assistant surgeon at the Children's Hospital, following which he became head of the bone and joint department of the Boston City Hospital, a post which he held from 1902 to 1931. He was chief-of-service at the Boston City Hospital from 1920 to 1931, and in 1927 organized the Bone and Joint Service. Upon resigning in 1931 he was elected president of the hospital staff. At this time he was also assistant professor of surgery at Tufts College Medical School and a lecturer on bone and joint surgery at Harvard Medical School and Harvard Medical School, Courses for Graduates.

Dr. Cotton was a founder and member of the board of governors of the American College of Surgeons. His affiliations included fellowships in the American Surgical Association, the American College of Surgeons, the Massachusetts Medical Society and the American Medical Association, and memberships in the Boston Surgical Society, the Boston Orthopedic Club and the New England Surgical Society. He was an honorary member of the American Academy of Orthopedic Surgeons.

His widow and a daughter survive him.

FISKE — EUSTACE L. FISKE, M.D., of 807 E. Windsor Road, Glendale, California, died March 20. He was in his seventy-ninth year.

Dr. Fiske was graduated from Harvard Medical School in 1886. He was a fellow of the American Medical Association and the Massachusetts Medical Society.

PIKE — FORREST W. PIKE, M.D., of Stoneham, died December 31, 1937. He was in his eightieth year.

He was a native of Stoneham and received his early education there. After receiving his degree from the University of Vermont College of Medicine, he served his internship in Poughkeepsie, New York, beginning private practice in North Chelmsford before finally settling in Portsmouth, New Hampshire. He practiced there for over thirty years, retiring twelve years ago.

Dr. Pike was a fellow of the Massachusetts Medical Society and a member of the American Medical Association. He was a member of the Masonic Order.

Four nieces and two nephews survive him.

NEW HAMPSHIRE MEDICAL SOCIETY

ONE HUNDRED AND FORTY-SEVENTH ANNUAL MEETING

HOTEL CARPENTER, MANCHESTER, N. H., TUESDAY AND WEDNESDAY, MAY 17-18, 1938

MONDAY, MAY 16

7:30 p. m. D.S.T.

HOUSE OF DELEGATES

Speaker, Richard W. Robinson, Laconia.
Vice-speaker, William J. P. Dye, Wolfeboro.

Delegates from New England Societies

Maine: Charles W. Kinghorn, Kittery.
Vermont: Sam Sparhawk, Burlington.
Massachusetts:
Daniel J. Ellison, Lowell.
Henry F. Dearborn, Lawrence.
Rhode Island:
Auray Fontaine, Woonsocket.
Francis H. Chafee, Providence.
Connecticut:
Thatcher W. Worthen, Hartford.
William E. Carroll, Meriden.

* * *

TUESDAY, MAY 17

9:30 a. m. D.S.T.

ROUND TABLE CONFERENCES

1. *Medicine: Symptoms and tests of the failing kidney.*
   Bruce Snow, Manchester.
2. *Surgery: Infections of the hand.*
   Clinton R. Mullins, Concord.
3. *Industrial Accidents.*
   Emery M. Fitch, Claremont.
4. *Orthopedics: The internist and his orthopedic problems.*
   Fred E. Clow, Wolfeboro.

11 a. m.

ROUND TABLE CONFERENCES

1. *Medicine: The simplification of laboratory diagnosis for the general practitioner.*
   Harold D. Levine, Bristol.
2. *Surgery: Sympathetic surgery in peripheral vascular disease.*
   M. Dawson Tyson, Hanover.
3. *Obstetrics: Work of Maternity and Infancy Committee.*
   Benjamin P. Burpee, Manchester.
4. *Dermatology: Treatment of common skin diseases.*
   J. Harper Blaisdell, Boston.

2:00 p. m.

Presentation of 50-Year Membership Gold Medals to:
Alpha H. Harriman, Laconia.
Frederick L. Hawkins, Meredith.
Henry O. Smith, Hudson.

Symposium on Thoracic Disease.

a. Treatment of Pneumonia. Frederick T. Lord, Boston.
Discussion opened by:
Francis H. Nolin, Claremont.
Harold D. Levine, Bristol.

b. Cancer of the Lung. Donald S. King, Boston.
Discussion opened by:
M. Dawson Tyson, Hanover.

c. Empyema. Edward D. Churchill, Boston.
Discussion opened by:
William J. P. Dye, Wolfeboro.

Surgical Diseases of the Extrahepatic Bile Ducts. I. S. Ravdin, Philadelphia.
Discussion opened by:
Walter H. Lacey, Keene.
Chester L. Smart, Laconia.

* * *

WEDNESDAY, MAY 18

9:30 a. m. D.S.T.

ROUND TABLE CONFERENCES

1. *Medicine: Diseases of the liver.*
   Leslie B. Copenhaver, North Woodstock.
2. *Surgery: The acute abdomen.*
   Herbert L. Taylor, Portsmouth.
3. *Ear, Nose and Throat: Every-day problems.*
   John A. Hunter, Dover.
4. *Pediatrics: Preventive medicine.*
   Ursula G. Sanders, Concord.

11:00 a. m.

Introduction of Visiting Delegates.

Blood Dyscrasias. J. H. J. Upham, Columbus, Ohio, *President,* American Medical Association.

2:00 p. m.

Report of House of Delegates.

Report of Trustees.

Diagnosis and Treatment of Intra-oral Cancer. Hayes E. Martin, New York City.

Discussion opened by:
George C. Wilkins, Manchester.

Treatment of Arthritis. Walter Bauer, Boston.

Discussion opened by:
Ezra A. Jones, Manchester.
Lawrence R. Hazzard, Portsmouth.

6:30 p. m.

THE BANQUET

Anniversary Chairman. Chester F. McGill, Portsmouth.

Introduction of President-elect.

Guest Speakers

Dr. Samuel T. Ladd, *president,* New Hampshire Medical Society.

Dr. J. H. J. Upham, *president,* American Medical Association.

Ladd's Lads

Manchester String Ensemble

BOARD OF REGISTRATION IN MEDICINE

Twenty candidates presented themselves at the meeting of the Board held on March 10. Eight candidates were permitted to take the written examination in which all were successful.

The annual directory of physicians licensed to practice in the State has been published. Copies may be obtained from Miss Harriet Parkhurst, State House, Concord, or from the secretary, Dr. Fred E. Clow, Brown House, Wolfeboro. The Board requests that physicians consult this list with a view to preventing practice by unlicensed persons. Recently a physician from a nearby state has maintained an office from which he has treated many patients, wholly without permission or legal authority. Infractions of the Medical Practice Act will be promptly reported to the county solicitors, once they are called to the attention of the Board or its investigator.

COMMITTEE ON CONTROL OF CANCER

The following letter has been mailed to all physicians in the state:

During and following the educational campaign of the Women's Field Army, you may be consulted by patients asking you to decide whether they have cancer. In order to assist you in such an examination, the following suggestive guide has been formulated:

1. Examine lips, buccal and pharyngeal membranes for ulcerations or nodules, and the cervical lymphatic areas for nodular enlargement.
2. Any patient with unexplained hoarseness, of over one week's duration, should be examined by an expert laryngologist.
3. Examine skin for new growths, bleeding ulcerations or scaly dry crusts. Black moles need especial consideration and expert advice.
4. Examine female breasts (and occasionally male) for lumps and for serous or bloody discharge from nipple.
5. Inquire minutely into symptoms of digestive disturbances, and if you cannot relieve them in a very short time, advise x-ray examination.
6. Examine carefully any patient with fairly rapidly increasing constipation, including digital rectal examination, stool examination for occult blood, and if indicated, an x-ray barium enema.
7. Rectal bleeding demands, first, digital examination; if no diagnosis can be made, then examine by proctoscopy. If no cause for bleeding is found with these two methods, then an x-ray barium enema should be advised. A barium enema should never be given until the rectum has at least been examined digitally.
8. Bimanual pelvic examination should be made in all women as a routine procedure. Note mobility, size and shape of uterus, appearance of cervix with especial attention to erosions. Note enlargement or tenderness of adnexa. If there is a history of abnormal bleeding, not definitely accounted for by the above examination, advise a diagnostic curettage.
9. If the patient has had urinary bleeding, advise immediate cystoscopy.
10. General examination should include the chest (if there is cough), abdominal palpation, and the examination of subcutaneous tissues and bones for possible tumors.

If the above rules are adhered to in making a physical examination, it is certain that fewer early cancers will be overlooked. Always remember that the fate of the cancer patient rests with the physician first consulted.

DISTRICT SOCIETY NEWS

Officers of the Belknap County Medical Society for 1938 are Dr. Harry Cheney, president; Dr. M. Alice Normandin, vice-president, and Dr. Clifford S. Abbott, secretary-treasurer. The next meeting will be held on April 12, with Dr. J. Russell Perley in charge of the program.

NOTES

Dr. George R. Eades, school physician, gave a lecture at a Parent-Teachers Association meeting at Keene on "Keeping the Child Well: Value of health clinics."

The quarterly meeting of the New Hampshire Graduate Nurses Association was held at the Elliot Community Hospital, Keene, under the presidency of Miss Ruth Whitcomb, of Concord, on March 15. The principal address was given by Mrs. Evangeline Morris, of the Simmons College faculty. Miss Marion Northway, of Milton, and Mrs. Melanie Proulx, of Manchester, were nominated for membership on the State Board of Nurse Examiners. Sectional meetings of public-health nurses, private-duty nurses and the League of Nursing Education were held in the morning. It was voted that the annual meeting be held in Manchester next June.

Dr. Sharples was guest-speaker at a recent meeting of the Whitefield Woman's Club. In a discussion of "Preventive Medicine" he gave a brief outline of the incidence of common diseases, and the work being done toward their prevention by private physicians and the federal, state and local health organizations.

MISCELLANY

MASSACHUSETTS TUBERCULOSIS LEAGUE

At the annual meeting of the Massachusetts Tuberculosis League, Incorporated, the following officers were elected for the ensuing year: Dr. Frederick T. Lord, Boston, president; Clifton H. Hobson, Palmer, and Dr. Francis P. Denny, Brookline, vice-presidents; Arthur Drinkwater, Cambridge, treasurer; Rodney Spring, Boston, assistant treasurer; Arthur J. Strawson, Newtonville, clerk. Dr. Henry D. Chadwick, the Rt. Rev. William Lawrence, Rabbi Harry Levi and Cardinal O'Connell were chosen honorary vice-presidents.

The meeting was followed by a talk by Dr. Lord on the prevention and control of tuberculosis in Massachusetts, and one by Dr. Robert E. Plunkett concerning the preventive research work in tuberculosis being carried out by the New York State Department of Health.

NOTES

At the fifth annual meeting of the American Institute of Nutrition, Dr. T. M. Carpenter, of the Carnegie Institution, Boston, was elected vice-president and Dr. Helen S. Mitchell, of Massachusetts State College, was made a member of the council.

Dr. G. Philip Grabfield, associate in pharmacology, Harvard Medical School, was elected secretary of the American Society of Pharmacology and Experimental Therapeutics at a recent meeting in Baltimore.

During the week beginning April 18 at the Peter Bent Brigham Hospital in Boston, Dr. Emil Goetsch, first resident surgeon, now professor of surgery at the Long Island Medical College, and Dr. Francis G. Blake, first of now-living resident physicians, at present Sterling Professor of Medicine, at Yale University School of Medicine, served respectively as surgeon-in-chief, pro tempore, and physician-in-chief, pro tempore.

CORRESPONDENCE

PERIODIC MEDICAL EXAMINATION OF ADULTS IN CONTACT WITH CHILDREN

To the Editor: Our attention has been called to an editorial under the title "An Opportunity for the District Medical Society" in your issue of March 24, in which you have commented favorably upon a plan for periodic medical examination of adults in contact with children that is being promoted by this Society.

Your comments in my opinion very faithfully interpret the underlying purpose of this work and we should be glad to assure the district medical societies through your good offices that it would be a pleasure for us to be of service to any of those societies which may desire to have more explicit information on the subject of this program.

JAMES BRYAN, *Executive Secretary,*
Medical Society of the County of Westchester.
171 East Post Road,
White Plains, New York.

MEDICINE IN COLONIAL MASSACHUSETTS

To the Editor: The following papers serve to throw a modest side light on the practice of medicine in the colony of the Massachusetts Bay.

HAROLD BOWDITCH, M.D.
520 Commonwealth Avenue,
Boston.

* * *

1

Boston 1715

The Estate of Mr James Butler
to Robert Ellis is Dr Viz:

By Former Acct. carried in	£ 2. 5. 0
For medicins and Attendance in time of his last sickness	£ 1. 3. 2
	£ 3. 8. 2

(Receipted:) Robert Ellis

2

The Estate of Mr. James Butler Deceasd
to Zab: Boylston is — Dr

To Medicine Adminesd 10th Attendance yor family from Time to time from 1711 April 12th to Decr 1713 in practice & shop	£ 3. 6.11
To Reducing a fracture ye Childs Choller bone & Cure	£ 0.15.—
	£ 4:01:11

Boston May 17th 1716

(Receipted:) Rec'd of Capt. Wm Bowditch & Mr Jno Eustus Adminestrs to ye Estate of Mr James Butler Deceas'd the Sum of four pounds one Shilling & 11d and is in full

p Zab: Boylston

3

For: francis Hamlens Wife Dr.

1729: May: 10 to Drops & Ceracloath (?)	00- 5- 2
1730: to a Visit & Electuary	00- 7- 6
Decembr 3 to Emptr Matricie to Attendance & Sp. *p* fumig.	00- 4- 8
	£ 00 s18 d10

Francis Gahtman
Chyrg

(Receipted:) Francis Gahtman

4

1738 Capt. Joseph Bowditch to John Cabot Junr Dr

Febry 22 To Medicines & Attende on yr Maid to March 19th	£ 1. 1.—

1739

1739

May 25 To Medicines & Attendance on yr wife & Children to Janry 21st } 1.12. 6
Febry 14 To Terebinth Venet. ℥i 1/ 0. 1.—
2.14. 6

Salem March 17th 1739
Recd in full of ye Above Accot:
p John Cabot Junr

5

1741 Capt. Joseph Bowditch to Jno Cabot Dr

July 23nd To Medicines & Attende on yr Family to Decemr 8th 1742 } £ 2.19. 4

Recd in full of ye above
p John Cabot

6

1743 Capt. Joseph Bowditch to John Cabot. Dr

Aprl 4 To Medicines & Attende on yr Child at Several times to May 26th 1743 } £ 2.18. 4
1744 To ½ yd Cloth &c. 1.16. 4
4.14. 8

Errors Excepted p John Cabot

7

Joseph Bowditch Esqr to Eben: Putnam Dr.

To Visits & Medicine adminsd him from July 9th till Octr. 21. 1771 Three Pounds Three Shillings & Four Pence - - £ 3. 3. 4

Errs excepted
Eben Putnam

Recd ye Above in full p Eben Putnam

8

1772 Joseph Bowditch Esqr to David Mason
feb. 7 To the use of my Elictrical Machine &c. 0.12. 0

David Mason

Febry 27th Cash pd. Mason's Son 12/.

9

Dear Sir,

George is rather heavy to day from being broke of his rest last night — the Symptoms of the disorder however are entirely off, & I expect after a nights sleep to find him well —

Ichabod has passed thro' the easiest of either —

Benj.'s Symptoms come on last & he still feels more of them than the others — I expect he will have the largest crop of pustles, I am not however apprehensive at present of so many as to be troublesome —

Charlotte feels a little of ye Symptoms yet & will probably next to Benj. have the most pock —

Your's with Respect
M L

(Endorsed:) Docktor Little
Feb 7 1794

Notes

1. Robert Ellis, surgeon, was the son of a surgeon, Edward Ellis, and the father of two surgeons, Edward and Thomas Ellis. In 1677 Edward Ellis, his father, is styled "chirurgeon" and in 1695 Robert Ellis is styled "barber chirurgeon," but later he too is called "chirurgeon." He lived in Boston at the corner of Winter and Newbury (now Washington) streets. The date of his death may be approximated through the fact that his will was dated February 23, 1719 and proved April 18, 1720.[1]

2. Zabdiel Boylston (1679-1766), of Brookline, is too famous for having introduced inoculation against smallpox into America to receive more than passing mention here. In the spring of 1721 Boston was invaded by smallpox from Barbados; the Rev. Cotton Mather suggested to Dr. Boylston that the Eastern practice of inoculation be tried, and two days later (June 26, 1721) Dr. Boylston inoculated his first patient. Against the opposition of other physicians and of a thoroughly aroused mob, and at the risk of his life, he persevered, and probably inoculated about two hundred and forty persons in all; the mortality was reduced from 12 to 15 per cent to a little over 2 per cent.[2]

3. Francis Gahtman was born in Hamburg, Germany, during the 1670s, and was living as late as 1740.[3] He appears in Salem as early as August 24, 1702, on which date, "being presently bound out to sea in a voiage in the good sloop called *The Charles* whereof Capt. Peter Lawrence is present commander against ye French and Spaniards"; he assigned to Henry Sharp of Salem, painter, because of a debt of £44.7s.9d, his share of any prizes that may be taken by Capt. Lawrence, together with his clothes, medicines and surgical instruments.[4] In the summer of 1703 John Quelch, mate of the brigantine *Charles*, threw the captain, who was ill, overboard, and assumed command as a pirate, returning to Marblehead in May, 1704. It was rumored that he had been joined by Capt. Larrimore, of Cape Ann, who had previously been a respectable privateersman. When a group of pirates had been located on Cape Ann, an armed force was sent to catch them, and "the judges rode to Salem and got more information from Doctor Gatchiman [probably Gahtman], whom they sent to the Governor to make a report." The pirates were captured at the Isles of Shoals and duly hanged in Boston.[5] Dr. Gahtman's part did not end here, for we find: "1729 April 17. A petition of Francis Gahtman, of Salem, Chyrurgeon, showing, that by order of this Government, 1704, Capt. Larramore and others were sent to England to receive their tryals, as accessories to Quelch's pyracy, he was sent over with them, and in his passage was taken and carried captive into France and lost his cloathing, books, chyrurgeon's chest and instruments, to a considerable value, praying for such relief as the Court shall think proper. Referred to the next May session."[6] The year 1708 finds him back in Salem, for in that year he married Mrs. Lydia West,[7] and later records about him are all of a peaceable and unexciting character — we find him signing a paper drawn up at a meeting of the proprietors of the common lands in 1713-1714,[7] being paid for his share in building a stone wall along the Lynn line in 1723,[8] bleeding a patient who kept a diary — James Jeffry — in 1724,[9] and mentioned as a communicant of the First Church in Salem in 1734-1735.[10] His name has been regularly mis-spelled, but his two signatures on this receipted bill show its correct spelling. As to the items: "ceracloath" is apparently cerecloth or waxed cloth; "Emptr Matricie" is undoubtedly the emplastrum hystericum or *Mutter-Pflaster* of the Ger-

mans; the curious may learn its composition in Dr. Woyt's *Gazophylacium Medico-Physicum* (1751).

4, 5, 6. Dr. John Cabot was the son of John Cabot of Salem, merchant. He was born in Salem in 1704, took the degree of A.B. at Harvard in 1724 and that of A.M. there in 1727, was placed twenty-first in his college class of forty, and was engaged in the practice of medicine in Salem by 1729. He died in Salem in 1749 leaving a widow and two young children; the latter died in 1750 and 1759, so that he left no descendants.[4, 11, 12]

7. Ebenezer Putnam, originally of Danvers, received the degree of A.B. at Harvard College in 1739 and that of A.M. in due course; he was placed seventeenth in a class of thirty-two. By the year 1744 he was practising medicine in Salem, where he joined the First Church and was chosen presiding elder on the death of Judge Ropes. He and Dr. Edward Augustus Holyoke, both "loyalists of the passive sort," were men of high standing in the community. In 1760 both are found among the founders of the Social Library. He died in 1788.[5, 9, 13] His patient, Capt. Joseph Bowditch, was paralysed for the last nine years of his life; as he died in 1780 it seems likely that the date of Dr. Putnam's first visit, 9 July 1771, was that of the stroke.

8. Colonel David Mason, the owner of the electrical machine, was an important figure in Salem, and took a prominent part in the episode at the North Bridge in 1775, when Colonel Leslie was obliged to retreat to Marblehead without capturing the guns which Colonel Mason had been instrumental in obtaining for the patriot party. He was a student of electricity and a friend of Benjamin Franklin, although some twenty years his junior.[13] This is no doubt an early instance of the use of electricity for paralysis.

9. Moses Little took the degree of A.B. at Harvard in 1787 and that of A.M. in 1804; he died in 1811.[13] There is nothing to show that this Moses Little was a physician; but a letter from a correspondent in Salem, writing June 2, 1811, says: "Doctor Little is confined to his chamber & says that for sometime past he has expected to die, but did not think he should have fallen away so fast." The correspondence of dates makes it practically certain that we are dealing with the same man. He might have attended the medical school in Boston, for the first class was graduated from there in 1788; but the apprentice system, which Dr. Little no doubt followed, was long in coming to an end. The internal evidence shows that this letter was written to Capt. Ichabod Nichols, of Salem, concerning four of his children who were no doubt under Dr. Little's care for inoculation against smallpox. The ages of the children may be of interest: George was sixteen, Ichabod ten, Benjamin eight and Charlotte six. All recovered, and died at the following ages: George at eighty-seven, Ichabod at seventy-five, Benjamin at sixty-two and Charlotte at eighty-four.

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6. Felt, J. B.: *Annals of Salem*. Vol. 2. 663 pp. Salem: W. & S. B. Ives, 1849.
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11. Briggs, L. V.: *History and Genealogy of the Cabot Family, 1475-1927*. 887 pp. Boston: Charles E. Goodspeed & Co., 1927.
12. *Harvard University Quinquennial Catalogue of the Officers and Graduates, 1636-1930*. 1463 pp. Cambridge: Harvard University, 1930.
13. *The Essex Institute Historical Collections*. Vol. 48. 393 pp. Salem: Essex Institute, 1912.

REPORT OF MEETING

HARVARD MEDICAL SOCIETY

A meeting of the Harvard Medical Society was held at the Peter Bent Brigham Hospital Tuesday evening, January 25, Dr. S. Burt Wolbach presiding.

The surgical case was presented by Dr. James B. Blodgett. The patient, a fifty-five-year-old Russian salesman, was first seen in the outpatient department two and a half months previously, at which time he complained of an inguinal hernia and of a chronic cough, both having been present for fifteen years. An x-ray taken at that time revealed a substernal mass and a triangular shadow in the periphery of the left lung field. The latter shadow was interpreted as a classical infarct of the lung. An x-ray taken one month later showed the triangular shadow to be larger, and he was hospitalized. At that time it was learned that he had suffered from weakness for the eight months preceding entry, and had lost 10 lb. in weight. Physical examination revealed a substernal mass which moved with deglutition, a few rales in the left chest, and a right direct inguinal hernia. Roentgenograms showed an increase in the size of the shadow in the left chest, the edges of which were rounded, suggesting a neoplastic process. The basal metabolic rate was +66, the cholesterol 235 mg. per cent, and the white-cell count 14,000. In spite of the markedly elevated basal metabolic rate there was no clinical evidence of thyrotoxicosis. The thyroid gland was removed for histologic study and relief of pressure symptoms, which the patient had experienced for a short time.

Dr. Elliott C. Cutler, in commenting on the case, stated that no definite diagnosis of the mass in the left chest could be made, although metastatic carcinoma of the thyroid was a definite possibility. Dr. Wolbach described the histologic picture of the removed thyroid, which appeared to be an embryonal adenoma in some areas, and a fetal adenoma in others. Such glands are not to be considered as true adenomas, but as a condition of compensatory hyperplasia. It is unusual for such a type of gland to give rise to metastases.

The second case was from the medical service. The patient, a twenty-eight-year-old, white housewife, suffered the sudden onset of transverse myelitis one month previously and was immediately admitted to the hospital. The past history was essentially negative. She had been married eight years, and had a seven-year-old child. Serological tests on both the husband and child were negative. Six months before entry to the hospital she consulted her local physician because of a persistent skin rash of one and a half years' duration. Her physician found that during a routine examination at the age of fourteen years a positive Wassermann reaction and a negative Hinton test were discovered. No therapy was given at that time. Similar results (positive Wassermann and doubtful Hinton) were reported by the hospital at the time of her delivery seven years previously, but the serological tests were not repeated and no therapy was given at that time. Her local physician found that the Wassermann reaction was negative, and the Hinton test doubtful, and believed

that the skin lesions were due to syphilis or tuberculosis. He administered intramuscular bismuth with marked improvement of the skin lesions, and advised a lumbar puncture which was refused by the patient.

Two weeks before her entry to the hospital the patient consulted another physician who found a doubtful Hinton test and gave her an injection of arsphenamine in an attempt to produce a "provocative reaction." A second injection of arsphenamine was given one week later, and was followed by a shaking chill, three days of asthenia, and the sudden onset of weakness and collapse on the fourth day with complete loss of motor and sensory function in both legs and incontinence of urine and feces. She was immediately admitted to the hospital.

At the time of entry she was incontinent, her bladder was distended, the abdominal reflexes were absent, the knee jerks were markedly diminished, the Babinski reaction was present bilaterally, and there was ankle clonus. Sensation was absent below the buttocks posteriorly, and the groins anteriorly. Ten days after entry the reflexes of the legs were hyperactive, and the Babinskis were still positive bilaterally. Two lumbar punctures yielded bloody fluid, thought to be due to trauma; the Wassermann reactions on both samples were negative. Four specimens of blood were taken. The Hinton test was doubtful in all four, while the Wassermann was positive in the first three and negative in the last. Tidal drainage of the bladder was instituted, and general supportive measures followed. There had been marked improvement, with return of most sensory and a great deal of the motor function. The knee jerks were still hyperactive, and the Babinskis were still positive.

Dr. C. Sidney Burwell stated that although the time relation between the injections of the arsphenamine and the transverse myelitis was impressive the mechanism of the production of the lesion was not understood. The arsenic may have produced its effect by direct action on the spinal cord, or by causing in an area of syphilitic inflammation an acute edema with partial or complete arteriolar occlusion and secondary spinal-cord change. Arsenic may also cause acute damage to the arteriolar wall itself, and hemorrhagic lesions are occasionally produced. In addition to transverse myelitis, acute hemorrhagic encephalitis may follow the administration of arsenicals. The prognosis in the present case was believed to be favorable.

The remainder of the evening's program was presented by Drs. Richard P. Strong, Henry Pinkerton and David Weinman who spoke on "Medical Investigations in Peru in 1937." Dr. Strong briefly outlined the historical development of our knowledge of the etiology of yellow fever, and the mechanism of its transmission.

Sanarelli in 1897 reported that an organism, *Bacillus icteroides,* caused the disease, and although this claim was confirmed by several investigators in the United States, it was later disproved. In 1900, Reed, Carrol, Lazear and Agramonte established the fact that the infective agent was present in the blood and that yellow fever is transmitted by the mosquito, *Aedes aegypti*. Although subsequent work (1905) showed that the virus was filterable, in 1909 Seidelin reported in the red cells an organism which he named *Paraplasma flavigenum*. Tyzzer, Sellards, and Dr. Strong were unable to find these structures in the red cells when they studied yellow fever in Guayaquil in 1913. Also, they were unable to cultivate any organism from the blood though they used Noguchi's spirochetal media of that time. Noguchi, however, also working in Guayaquil, believed that he had found the causative organism in 1918 when he demonstrated and cultivated *Leptospira icteroides* from guinea pigs inoculated with the blood of jaundiced patients. Later investigations showed that this organism was identical with that causing epidemic jaundice (Weil's disease), and that perhaps the patients used by Noguchi were suffering from the latter disease and not yellow fever, or that the guinea pigs were infected with hemorrhagic jaundice. The present belief is that yellow fever is caused by a filterable virus.

Until 1932 yellow fever was believed to be especially a coastal disease and to affect particularly the population of our large seaports. Its favorable haunts were squalid low-lying areas with open drainage systems and heavy mosquito infestation. It was thought to be essentially a mosquito-borne disease. Rarely was it known to extend far inland or to ascend high altitudes.

However, in 1932 Soper and his associates of the Rockefeller Foundation called attention to the discovery of yellow fever in Brazil and elsewhere in South America, in rural areas or sparsely populated jungle districts, and, importantly, in the absence of *A. aegypti*. A number of outbreaks have been reported in the past few years, and a few at altitudes of 2000 feet or over.

While in South America in 1937, Hanson and Dr. Strong were asked to investigate an epidemic that was occurring on a coffee plantation on the eastern slopes of the Andes, at an elevation of 2500 feet. The nature of the epidemic was unknown. A study of the epidemic was made and an autopsy on a fatal case revealed the typical saffron-ocher liver with slight nutmeg mottling, the hemorrhagic gastritis with clots of dark blood in the stomach and duodenum, and the acute parenchymatous nephritis, which, together with the subsequent study of sections of the organs, established the diagnosis of yellow fever. Diligent search failed to reveal the presence of *A. aegypti* or other possible insect vectors for the disease. No explanation of the mode of transmission of the disease in inland regions has yet been found, though it is a problem which the Rockefeller Foundation is still studying.

The expedition to Peru in 1937, as was the expedition of 1913, was organized for the purpose of studying Oroya fever or verruga peruviana. This disease was referred to over four centuries ago when the Portuguese and Spanish invaders of Central and South America were said to be afflicted with it, and to suffer a high mortality. De la Vega stated that one quarter of Pizarro's forces succumbed to its ravages. The disease is characterized by fever, anemia and an eruption upon the skin. Often two stages are observed: in the first, Oroya fever, there is fever and marked anemia; in the second, verrugous lesions appear; sometimes only the second stage is apparent. If the patient survives the first stage of the disease, later a miliary eruption may occur, and subcutaneous, cherry-red, easily bleeding nodules develop, which may later become 7 or 8 cm. in diameter and tend to become eroded.

Barton in 1909 described bodies in the red blood corpuscles which he thought were probably the cause of the disease. In 1913, Tyzzer, Sellards and Dr. Strong observed these bodies, and after studying them, believed they were parasitic; in honor of Barton, they named the organism *Bartonella bacilliformis*. The organism is rod-shaped or rounded. The rods measure 1.5 to 2.5μ in length, and the rounded form 0.5 to 1μ in diameter. The anemia is frequently very severe and fatal. The red blood cells may be less than 1,000,000 per cubic millimeter. In severe cases over 90 per cent of the red cells are invaded by the parasite. The anemia is macrocytic in type, with marked anisocytosis and poikilocytosis, and the appearance of normoblasts and megaloblasts.

In 1913 attempts to cultivate this organism, especially on Noguchi's spirochetal media of this period, were unsuccessful. However, Dr. Strong and his associates were able to inoculate monkeys successfully with material from human verruga nodules, and to reproduce the characteristic nodules through a long series of these animals. A monkey and two rabbits inoculated intravenously with blood from a human case of Oroya fever with anemia, containing large numbers of *B. bacilliformis* in the red cells, did not develop any verrugas, nor were other lesions found in them. Also, a man, who volunteered, was inoculated cutaneously with a human verruga nodule, and did not develop fever, anemia or *B. bacilliformis* in the blood, but only a local verruga lesion. The results of such experimental work obviously did not demonstrate or permit the assumption upon a scientific basis that the pathologic processes involved in Oroya fever and the verruga stage of the disease were identical.

However, by 1926, Noguchi had greatly improved his leptospiral media which he had also used for the cultivation of protozoa, and in this year he was able to cultivate *B. bacilliformis* from blood of Oroya fever cases sent to him in New York. Battistini was associated in this work. On the expedition of 1937, using this same media, Dr. Strong and his associates were able to confirm and extend this work.

The disease is found only in regions lying between 2400 and 8000 feet in elevation, where the climatic conditions are peculiar — receiving little rain, and situated along the banks of small rivers. While the method of transmission has not been demonstrated there is some evidence which indicates that Phlebotomus, a small blood-sucking sand fly, may be concerned in the transmission. Both Townsend and Shannon and several others have been in favor of the view of such transmission, and Shannon has recently made a very valuable epidemiological contribution to the subject. Dr. Hertig is at present in Peru carrying on further observations in this connection.

Dr. Pinkerton described the pathologic lesions of verruga peruviana, and the cultural characteristics of the Bartonellae. Histologically the verrugous lesions appear to be multiple rapidly growing hemangiomas, somewhat resembling the hemorrhagic sarcoma of Kaposi. Bartonellae can be demonstrated in the endothelial cells during the verrugous stage, and appear to be the cause of the tumors. Cutaneous lesions were produced in monkeys by using Bartonellae isolated from human cases suffering from the febrile stage, and cultured on Noguchi's medium. This suggested that the two stages of the disease, verruga peruviana, had a similar etiology. A comparative study of the viruses isolated from the acute febrile and the verrugous stages gave further support to the theory that both have a common etiology. As cultured in tissue culture both viruses had identical growth patterns within cells. Unlike the Rickettsiae, which grow only intracellularly, the Bartonellae will grow outside the cells of the tissue culture as well as within cells. The morphology of the Bartonellae as demonstrated by special staining methods in the tissue of cases of the febrile and verrugous stages was also found to be identical.

Dr. Weinman described the characters of the Bartonellae, and the production of the various forms of Carrion's disease in monkeys. The verrugous and the anemic forms were both produced in *Macacus rhesus* monkeys by injecting organisms obtained directly from human beings with the anemic form of the disease; splenectomy prior to inoculation greatly facilitated the productions of the anemic form. Nine per cent of the persons studied in a Peruvian village where Carrion's disease is endemic were found to be infected with Bartonellae, although they were without symptoms. Some of these individuals had never suffered from the disease; others gave a history of previous illness. This infected portion of the population may serve as a reservoir for the disease. Experimental asymptomatic infection of the monkey was also obtained. Peruvian guinea pigs from the endemic regions were found to be infected with Bartonellae, although investigation showed that these organisms were different in type from the human form. The natural means of transmission of the disease still remains undetermined.

NOTICES

HENRY JACKSON LECTURE

The Henry Jackson Lecture for 1938 offered by the New England Heart Association will be given by Louis Hamman, M.D., associate professor of clinical medicine, Johns Hopkins University School of Medicine, at 8:15 p. m. on Friday, April 29, at the Boston Medical Library. His subject will be "The Diagnosis of the Causes of Heart Failure."

The annual business meeting of the New England Heart Association will precede the lecture.

RADIO BROADCASTS

The eighth group of weekly broadcasts sponsored by the American Medical Association and the National Broadcasting Company concern hygiene. These dramatized health messages are intended to furnish supplementary material for health teaching in junior and senior high schools and are broadcast every Wednesday from 2:00 to 2:30 p. m. over the Red Network. The dates and subjects are as follows:

April 27 — Healthier Babies. Daily routine of the healthy baby; medical supervision; feeding.

May 4 — Healthier Mothers. General advice for the expectant mother; good for boys and girls to know about.

May 11 — Hospitals Aid Health. The place of the hospital in the health program of the individual and the community.

May 18 — Runabouts, 1938 Model. The preschool child and the health and personality problems of that age.

CANCER CLINIC

A cured-case teaching cancer clinic will be held at the Boston Dispensary on Friday, April 29, at 11:30 a. m., at which time a variety of cured cases will be shown and methods of treatment discussed.

Physicians are cordially invited to attend.

CAMBRIDGE HOSPITAL

The regular clinicopathological meeting of the staff of the Cambridge Hospital will be held at the Cambridge Hospital, 330 Mt. Auburn Street, on Tuesday, April 26, at 8:30 p. m.

Dr. Stanley Nowak and Dr. Stephen Maddock will discuss sudden death in liver disease.

All members of the medical profession are cordially invited to attend.

JOSEPH M. WADDEN, M.D., *Secretary*.

CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS, UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

Clinic	Date	Orthopedic Consultant
Salem	May 2	Harold C. Bean
Lowell	May 6	Albert H. Brewster
Gardner	May 10	Mark H. Rogers
Haverhill	May 11	Arthur T. Legg
Brockton	May 12	George W. Van Gorder
Pittsfield	May 16	Francis A. Slowick
Springfield	May 18	Garry deN. Hough, Jr.
Worcester	May 20	John W. O'Meara
Fall River	May 23	Eugene A. McCarthy
Hyannis	May 24	Paul L. Norton

MASSACHUSETTS MEMORIAL HOSPITALS

The annual reunion of the House Officers Alumni Association of the Massachusetts Memorial Hospitals will be held on Saturday, April 23. The morning will be devoted to clinics by the medical, surgical, pediatric, obstetrical and outpatient departments and the staff of the Evans Memorial. These will start at 10:00, and will be followed at 12:30 by a luncheon and a business meeting.

The following papers by members of the staff will be read in the afternoon:

2:00-2:20. Two Cases of Leg Lengthening.
Dr. Daniel M. Killoran.

2:20-2:40. Comparative Results with Different Methods of Handling the Appendiceal Stump.
Dr. Welman B. Christie.

2:40-3:00. Carcinoma of the Colon.
Dr. Clifford D. Harvey.

3:00-3:20. Osteomyelitis of the Frontal Bone.
Dr. Leighton F. Johnson.

3:20-3:40. A Fifteen-Year Calculus Survey of the Massachusetts Memorial Hospitals.
Dr. Myron J. Hahn.

3:40-4:00. Recent Advances in the Treatment of Cancer.
Dr. Thomas J. Anglem.

4:00-4:20. Radiation of Inflammatory Lesions.
Dr. George Levene.

4:20-4:40. Parathyroid Tumors.
Dr. Louis G. Howard.

Cocktails will be served at the Hotel Kenmore from 5:30 to 7:00, to be followed by the annual banquet.

THE MASSACHUSETTS PUBLIC HEALTH ASSOCIATION

The April meeting of the Massachusetts Public Health Association and its sections will be held on Wednesday, April 27, at the University Club, 40 Trinity Place, Boston.

The section meetings will start at 11:00 a. m. Luncheon will be served at 1:00 p. m., after which Dr. Cecil K. Drinker will speak on "The Spread of Infections in the Respiratory Tract."

The programs for the different sections are as follows:

BOARD OF HEALTH SECTION

The Preparation and Scope of a Municipal Sanitary Code. Dr. Murray P. Horwood.

LABORATORY SECTION

The Laboratory Diagnosis of Virus Diseases. Dr. Sidney C. Dalrymple.

New Developments in Milk Technology. Dr. Robert E. Bemis, and Mr. A. A. Robertson.

Movies on Bacteriological Technic. Dr. Alice T. Marston.

CHILD HYGIENE AND PUBLIC HEALTH NURSING SECTION

Business meeting.

The Outlook for an Appraisement of Nutrition. Dr. Harold Stuart.

Ernest M. Morris, M.D., *President*,
G. Donald Buckner, *Secretary*.

AMERICAN HEART ASSOCIATION

The fourteenth scientific session of the American Heart Association will be held on June 10 and 11 from 9:30 a. m. to 5:30 p. m. in the Sir Francis Drake Hotel, San Francisco, California.

On Friday, June 10, the general heart program will be given, and on Saturday, June 11, the program of the Section for the Study of the Peripheral Circulation.

NEW ENGLAND HEART ASSOCIATION

The next meeting of the New England Heart Association will be held at the Boston Medical Library on Monday, April 25, at 8:15 p. m.

PROGRAM

Heart Disease in Pregnancy at the Worcester City Hospital. Dr. F. B. Carr, of Worcester.

Electrocardiographic Changes in Vitamin B Deficiency. Drs. C. C. Dustin, H. L. C. Weyler and C. P. Roberts, of Providence, Rhode Island.

Gall-Bladder Disease and Coronary Sclerosis. Dr. E. H. Drake, of Portland, Maine.

A Case of Hemophilia with Hemopericardium as a Result of Trauma. Dr. C. C. Dustin, of Providence, Rhode Island.

Clinical Observations on the Use of Quinidine. Dr. James Z. Naurison, of Springfield.

Auricular Flutter: Report of an unusual case with some remarks about the history of our knowledge of this disorder. Dr. Frank T. Fulton, of Providence, Rhode Island.

Interested physicians and medical students are invited to attend.

James M. Faulkner, M.D., *Secretary*.

COURSE IN OCCUPATIONAL DERMATOSES

During May, 1938, a course in occupational dermatoses will be given under the auspices of the Harvard School of Public Health. Lectures will be given on the clinical manifestations, aspects, and so forth. Clinics will be held at the Massachusetts General Hospital, and at the offices of various insurance companies. Visits to some of the more important factories will be arranged, so that students may study industrial processes and preventive measures. The number of students will be limited to ten. Further information and registration blanks may be obtained from the Dean of the Harvard School of Public Health, 25 Shattuck Street, Boston, Mass.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, APRIL 25

MONDAY, APRIL 25

8:15 p. m. New England Heart Association. Boston Medical Library, 8 Fenway.

TUESDAY, APRIL 26

*9-10 a. m. Boston Dispensary. Clinicopathological conference. Dr. R. C. Wadsworth and Dr. Cadis Phipps.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

8:30 p. m. Clinicopathological meeting. Cambridge Hospital.

WEDNESDAY, APRIL 27

*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.

11 a. m. Massachusetts Public Health Association. University Club, Boston.

*12 m. Clinicopathological conference. Children's Hospital amphitheater.

THURSDAY, APRIL 28

8:30-9:30 a. m. Exchange visit, surgical and orthopedic staffs of the Peter Bent Brigham and Children's hospitals, held this week at the Children's Hospital. *This is the last exercise for the season.*

*9-10 a. m. Boston Dispensary. The Significance of the Heterophile Agglutination Test. Dr. Peter Beer.

FRIDAY, APRIL 29

*9-10 a. m. Boston Dispensary. Social Security. Professor E. B. Wilson.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

*11:30 a. m. Cancer clinic. Boston Dispensary.

12 m. Clinical meeting of the Children's Medical Service, Massachusetts General Hospital, Ether Dome.

8:15 p. m. Henry Jackson Lecture, Boston Medical Library, 8 Fenway.

SATURDAY, APRIL 30

*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. Thannhauser.

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

SUNDAY, MAY 1

4 p. m. Illustrated, public health lecture, Faulkner Hospital auditorium. Progress in Dental Surgery. Dr. Kurt H. Thoma.

*Open to the medical profession.

APRIL 22 — Symposium in honor of Dr. Jelliffe. Page 583, issue of March 31.

APRIL 22 — Massachusetts Society for Social Hygiene. Page 658, issue of April 14.

APRIL 23 — Massachusetts Memorial Hospitals, annual reunion of house officers' alumni association. Page 707.

APRIL 23 — Symposium on pulmonary disease. Boston City Hospital. Page 659, issue of April 14.

APRIL 25 — New England Heart Association. Page 707.

APRIL 26 — Lawrence Cancer Clinic. Page 658, issue of April 14.

APRIL 26 — New England Society of Psychiatry. Page 322, issue of February 17.

APRIL 26 — Clinicopathological meeting. Cambridge Hospital. Page 706.

APRIL 27 — Massachusetts Public Health Association. Page 707.

APRIL 29 — Cancer clinic. Page 706.

APRIL 29 — Henry Jackson Lecture. Boston Medical Library. Page 706.

MAY 12 — Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, 8:30 p. m.

MAY 16 and 17 — American Neisserian Medical Society. Page 582, issue of March 31.

MAY 31, JUNE 1 and 2 — Annual meeting of the Massachusetts Medical Society. Hotel Bradford, Boston.

JUNE 6, 7, 8, and 9 — American Association of Industrial Physicians. Page 499, issue of March 17.

JUNE 10 and 11 — American Heart Association. Page 707.

JUNE 13-17 — American Medical Association. San Francisco.

JUNE 13, OCTOBER 8 and NOVEMBER 15 — American Board of Ophthalmology. Page 282, issue of February 10.

SEPTEMBER 12-14 — American Association for the Study of Goiter. Page 545, issue of March 24.

OCTOBER 17-21 — Clinical Congress of the American College of Surgeons, New York City.

OCTOBER 24-26 — Academy of Physical Medicine, Scientific Session. Washington, D. C.

DISTRICT MEDICAL SOCIETIES

BRISTOL SOUTH

MAY 5 — 5 p. m., New Bedford.

ESSEX SOUTH

MAY 5 — Censors meet at Salem Hospital, 3:30 p. m.

MAY 11 — Annual meeting, Salem Country Club, Peabody. Dinner at 7 p. m. Speaker and subject to be announced.

FRANKLIN

Meeting will be held at the Franklin County Hospital, Greenfield, at 11 a. m. the second Tuesday of May.

HAMPDEN

Meetings will be held on the fourth Tuesday in April and July.

HAMPSHIRE

MAY 11 — Page 546, issue of March 24.

MIDDLESEX EAST

Meeting will be held at the Bear Hill Golf Club, Stoneham, at 12:15 p. m. on May 11.

MIDDLESEX NORTH

Meeting will be held at the Vesper Country Club, Lowell, on April 27.

NORFOLK DISTRICT

MAY — Annual meeting.

The censors meet on the first Thursdays of May and November in each year.

NORFOLK SOUTH

MAY 5 — Annual meeting, at 12 noon.

PLYMOUTH

Meetings will be held at 11 a. m. on April 21, May 19 and July 21.

WORCESTER

MAY 11 — Afternoon and evening, annual meeting. Place and schedule of program to be announced.

BOOKS RECEIVED FOR REVIEW

Handbook on Social Hygiene. Edited by W. Bayard Long and Jacob A. Goldberg. 442 pp. Philadelphia: Lea & Febiger, 1938. $4.00.

Treatment in General Practice. Harry Beckman. Third edition. 787 pp. Philadelphia and London: W. B. Saunders Company, 1938. $10.00.

How Ancient Healing Governs Modern Therapeutics: The contribution of Hellenic science to modern medicine and scientific progress. Kleanthes A. Ligeros. 523 pp. New York and London: G. P. Putnam's Sons, 1937. $10.00.

Introduction to Ophthalmology. Peter C. Kronfeld. 331 pp. Springfield, Illinois and Baltimore: Charles C Thomas, 1938. $3.50.

The Thousand Forms of Disease. R. P. Byers. 29 pp. Boston: Superuniversity Publications, 1938. $1.50.

The Biology of Pneumococcus: The bacteriological, biochemical and immunological characters and activities of Diplococcus pneumoniae. Benjamin White. 799 pp. New York: The Commonwealth Fund, 1938. $4.50.

The Patient and the Weather. William F. Petersen, with the assistance of Margaret E. Milliken. Volume 4, part 3. 651 pp. Ann Arbor: Edwards Brothers, Inc., 1938. $10.00.

Physicians' Vitamin Reference Book: Presenting to the clinician a useful compendium of the latest facts about vitamins. Medical Division, Professional Service Department, E. R. Squibb & Sons. 126 pp. New York: E. R. Squibb & Sons, 1938.

Illness: Its story and some common symptoms, a guide for the layman. S. Henning Belfrage. 173 pp. New York: Oxford University Press, 1938. $1.50.

Pulmonary Tuberculosis in Practice: A modern concep-

tion. R. C. Wingfield. 122 pp. Baltimore: William Wood & Company, 1937. $2.50.

Fractures and Dislocations for Practitioners. Edwin O. Geckeler. 252 pp. Baltimore: William Wood & Company, 1937. $4.00.

Step by Step in Sex Education. Edith Hale Swift. 207 pp. New York: The Macmillan Company, 1938. $2.00.

X-Rays and Radium in the Treatment of Diseases of the Skin. George M. MacKee. Third edition, thoroughly revised. 830 pp. Philadelphia: Lea & Febiger, 1938. $10.00.

Scientific Contributions in Honor of Joseph Hersey Pratt on His Sixty-Fifth Birthday. Anniversary Volume. By his friends. 983 pp. Lancaster: Lancaster Press, Inc., 1937.

BOOK REVIEWS

Surgical Pathology of the Diseases of the Neck. Arthur E. Hertzler. 237 pp. Philadelphia, Montreal and London: J. B. Lippincott Company, 1937. $5.00.

This work is the tenth of Hertzler's monographs on surgical pathology. In the preface to this volume the author deplores "the present tendency to treat all lesions of the neck by irradiation without a definite pathologic diagnosis" and adds "what surgeons have learned is in grave danger of being lost by the practice."

He proceeds in a clear, concise and orderly manner to discuss not only the gross and microscopic pathology of the diseases of the neck but also their life history. The study is, therefore, of great practical value to the clinician and surgeon in the differential diagnosis of these conditions. To this end the many excellent illustrations are of valuable assistance. The pages carry 206 illustrations. Ninety-eight are pictures of patients showing the early and progressive appearances of the various diseases. The remaining cuts (except two roentgenograms of the chest) illustrate gross and microscopic pathology.

The subject is discussed under ten chapter headings; preview of surgical affections, Hodgkin's granuloma, lymphosarcoma, lymphoepithelioma, rare primary tumors, vestigial rests, benign tumors, salivary tumors, secondary tumors and inflammatory affections. (Hertzler has dealt with diseases of the thyroid gland in a previous monograph.) Following each chapter is a short bibliography with appraisals by the author. The text is often colored by picturesque expressions.

The physical make-up of the volume is attractive. The paper is highly calendered. The typography is excellent. The book merits a place on the shelves of every general surgeon.

Pathology of the Central Nervous System. Cyril B. Courville. 344 pp. Mountain View, California: Pacific Press Publishing Association, 1937.

This book adequately covers the subject and, moreover, gives many new, fresh points of view in regard to the pathology of the nervous system. The author has been fortunate in having had at hand for statistical data and examination the brains and spinal cords in a series of 15,000 autopsies performed at the Los Angeles County Hospital. His book is the result of his labors in this field.

The usual aspects of the subject are covered: congenital anomalies, malformations, diseases of the blood vessels, infectious diseases, trauma, intoxications and tumors. Two chapters are worthy of special comment; the one on the infectious diseases of the nervous system covers the field more adequately than any other textbook, and the same may be said about the section on trauma. A series of excellent illustrations of pathologic material, both gross and microscopic, plus many diagrams and drawings, illustrates the volume. Particularly useful are the drawings in the chapter on trauma, showing the mechanism of injuries to the nervous system, and those delineating the sources of metastatic tumors from other organs. As an appendix there is an unusual chapter on clinicopathological aphorisms, and the book closes with an adequate index.

Printed on rather heavy, shiny paper, the book is not particularly attractive typographically. The matter in it, however, is the product of a thoughtful, ingenious, painstaking individual and deserves wide reading. Although there have been a number of books on the pathology of the nervous system published in recent years, this volume, unique in character, deserves a place in every neurologist's library. The subject is, perhaps, a little oversimplified for the neurological expert, but for students it offers a splendid text.

Macleod's Physiology in Modern Medicine. Edited by Philip Bard et al. Eighth edition. 1051 pp. St. Louis: The C. V. Mosby Company, 1938. $8.50.

This book appears in a new eighth edition, revised and largely rewritten by Prof. Philip Bard, of Johns Hopkins, with the help of eight collaborators, leaders in special physiological fields. In this edition, the section on the physicochemical basis of physiologic processes has been omitted, and the book as a whole contains less physiologic chemistry, allowing room for the expansion of other material. A satisfactory arrangement is the division into large and small type, some of the clinical applications, methods and more technical considerations being given in the small print.

From the clinical point of view, one notes that brief mention only is given the new precordial lead in electrocardiography. The old terms for right and left bundle-branch block are used, while the newer and exactly opposite interpretation, now generally accepted by clinicians, is only mentioned as a possibility.

This is not primarily a book on applied physiology, but a broadly based textbook. Its suitability, however, is by no means limited to medical students. Any practitioner, and particularly any clinician who pretends to master the field of his special interest, will find fundamental physiology brought up to date, together with adequate references to a further perusal of the literature.

The mutual dependence and indebtedness which exists between the so-called fundamental sciences and their clinical application is happily being recognized by both physiologists and practicing doctors. Prof. Henry C. Bazett closes the section on the circulation with the following words:

> Physiology has something to contribute to medicine, particularly, perhaps, a scientific attitude, a meticulous attention to detail, and a more quantitative approach to the subject. But the contribution of medicine to physiology is no less important; for scientific medicine has contributed largely to the solution of the problem of vascular control in inflammation, to the study of excitation and conduction in the heart, and many other problems, sometimes in meticulous detail, sometimes in large outline of basic fundamental principles. When the pressing problem of hypertension is solved, it is as likely to be solved by the clinicians and to throw light on physiology, as it is to be solved by the physiologist and to throw light on medicine.

Health Under the "El": The story of the Bellevue-Yorkville Health Demonstration in mid-town New York. C.-E. A. Winslow and Savel Zimand. 203 pp. New York and London: Harper & Brothers, Publishers, 1937. $2.25.

In this book with illustrations, charts and tables, Winslow and Zimand describe the health demonstration that was conducted in the Bellevue-Yorkville district of New York City over a ten-year period. The area of this district was approximately 9 per cent that of the entire city, and the permanent population comprised a similar proportion. During the day a large influx of individuals from other localities came into the district and were subjected to some of the health hazards of the permanent residents.

The demonstration was financed in part by the existing agencies and in part by a grant from the Milbank Foundation.

The objectives of the demonstration were:

1. To apply to a given area known facts about the prevention of disease.
2. To interest the district in the improvement of its health.
3. To further develop, by careful analysis and research, methods of public-health administration that are practical and useful in a city of the first class.
4. To supplement existing health agencies, both public and private, to such an extent as to make their facilities reasonably adequate to meet the needs of the population. This implies a health program for the district that, if successful, may be applicable to the whole city and to other urban areas.
5. To integrate the work of the demonstration so thoroughly with the Health Department and other agencies that the gains of the demonstration will be conserved after the demonstration itself is completed.

At the close of the ten-year period, substantial improvements in the health conditions of the district were noted. The tuberculosis rate had dropped, the typhoid rate had dropped, and no diphtheria cases occurred in 1933. Infant mortality showed a marked decline.

The practicing physician was considered in planning the demonstration. Dr. Charles G. Heyd said: "It seems to me in a far larger sense the usefulness of the Bellevue-Yorkville demonstration has been that it is the first concrete example of the introduction of the medical profession into an ideal set-up for health service."

Success of the projects planned to stimulate and aid in the practice of truly preventive medicine of the private physician was less complete than had been hoped. The graduate course offered to physicians in the technic of making general physical examinations was discontinued. The service of the contact worker who visited physicians in their offices to discuss with them ways of increasing their preventive practice was also discontinued. The general practitioners did not make use of laboratories which were made available to them to any great extent. Nurses were furnished to physicians to help them in furthering preventive work. Only a few physicians availed themselves of this service.

Winslow and Zimand state:

> What triumphed at Bellevue-Yorkville was not merely an administrative technic, but an ideal, and an ideal by no means limited to the field of health. It was the basic ideal of democratic government.
>
> In one sense, the Bellevue-Yorkville demonstration was impermanent. Already, external evidences of its existence are passing. . . . The personnel has been scattered, the building may be converted to other uses. In its technical details, too, the work will pass.
>
> In another sense, however, the work is lasting. Bellevue-Yorkville was not, after all, concerned mainly with introducing specific types of public-health work, with demonstrating the value of a particular method of procedure, a type of equipment. Its function was more basic. These changing, impermanent features were only the tools with which it worked — the means it used to carry out its fundamental purpose, which was, in its essence, an educational process for attaining an ideal of social co-operation.

One cannot help but wonder if this plan had taken into account to a far greater extent than it did the physicians in the district, — if it had been based more on the line of the Hunt plan, — if the results might not have been better.

Regardless of whether one is sympathetic with the undertaking, this book should be read by all students of medicosocial economic problems, as it offers one method toward their solution.

The Compleat Pediatrician: Practical, diagnostic, therapeutic and preventive pediatrics. Wilburt C. Davison. Second edition. 243 pp. Durham, North Carolina: Duke University Press, 1938. $4.00.

Here is a book small enough to carry easily in one's professional bag but which contains, nevertheless, about as much practical pediatric information as can be found in any book in the language. Included are discussions of some 329 diseases, 213 laboratory procedures and therapeutic technics, outlines of the principles of growth, development and child guidance and, for the less experienced, directions for history taking and the making of examinations.

But notwithstanding its comprehensiveness, its accuracy and its up-to-dateness this will not become every man's book. The arrangement of its matters is unusual and perhaps at first glance puzzling. And though no great effort is required to learn how to use it properly, there will be some (the reviewer is not including himself among the number) who will never learn to like it. Diseases are arranged in groups not on the basis of etiology but according to the anatomical system — respiratory, gastrointestinal, circulatory, neuropsychiatric, and so forth — through which their objective manifestations are most characteristically expressed. One in pursuit of a diagnosis proceeds from signs and symptoms to the disease and thence, it may be, to laboratory tests by which the diagnosis may be confirmed or rejected. In short, the approach is much the same as in using an index of differential diagnosis. If one be constitutionally inimical to such approach this book is emphatically not for him; if the method appeals he will come to value Dr. Davison's work very highly.

The potential user should be assured in any case, however, that this second edition, entirely rewritten, is much easier to manage than was the first; the paragraph numbers have been reduced from some 1100 to about one fifth that number, thus greatly facilitating the pursuit of cross-references.

In preparing this book the author drew on no less than 7500 articles in various medical journals, but in order not to encumber the text with additional numbers the bibliography has been separately printed. It may be obtained from the publishers for fifty cents and may be inserted in the book at the option of the reader.